JOURNAL
of the
American Veterinary Medical Association
Formerly AMERICAN VETERINARY REVIEW
(Original Official Organ U. S. Vet. Med. Assn.)
H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

R. S. MacKellar, Pres., New York, N. Y. M. Jacob, Treas., Knoxville, Tenn.

EXECUTIVE BOARD

Cassius Way, Member-at-Large and Chairman;
C. P. Fitch, ex officio; R. S. MacKellar, ex officio;
A. E. Cameron, 1st District; E. P. Althouse, 2nd District; L. A. Merillat, 3rd District;
C. A. Cary, 4th District; F. F. Parker, 5th District; L. M. Hurt, 6th District;
C. H. Hays, 7th District; J. C. Flynn, 8th District; H. W. Jakeman, 9th District; O. V. Brumley, 10th District.

The American Veterinary Medical Association is not responsible for views or statements published in the Journal, outside of its own authorized actions. Reprints should be ordered in advance. Prices will be sent upon application.

Vol. LXXXV, N. S. Vol. 38 NOVEMBER, 1934 No. 5

VETERINARY STUDENT ENROLLMENT FOR 1934-35

An increase of over 72 per cent in the number of freshmen students enrolled in the twelve veterinary colleges in the United States and Canada this fall, as compared with one year ago, has occasioned considerable surprise even upon the part of some of our veterinary educators. Practically without exception, whenever this situation is mentioned, we are asked to give the probable reason or reasons for this unusually large increase. We have become used to seeing each crop of freshmen students a little bit larger than the year before, but we have not had any such increase (from 331 to 572) in recent years.

The total number of students pursuing courses in our veterinary colleges this year exceeds, by 316, the number enrolled for the year 1933-34. More than 75 per cent of this increase is found in the larger number of freshmen registered this year as compared with a year ago. The balance of the increase is to be found in the number of students registered for the pre-veterinary year in the four colleges which report on such students. This year there are 222 in this classification. Although the totals for the sophomore and junior classes are less than the corresponding figures for a year ago, the number of senior students this year is the largest for any year since 1919-20.

It would be quite difficult, if not impossible, to determine definitely the reason for the interest being shown in veterinary medicine reflected in the increased enrollment of students in our veterinary colleges. The only way that this could be done with any degree of accuracy would be to find out from each one of the new students this year just why he or she selected veterinary medicine for a career. However, from the information at hand, it would appear that quite a number of factors have been operating.

First of all, it might be mentioned that the veterinary profession has been the subject of a considerable amount of newspaper publicity during the past couple of years, on one score or another. There have been a number of syndicated articles that have appeared in newspapers all over the country, directing attention to the possibilities of the veterinary profession. The matriculation of three women students at Ohio State University, two years ago, was the subject of one feature article that was published throughout the length and breadth of the land. The same thing happened one year ago, when the University of Pennsylvania Veterinary School opened its doors to the fair sex. Although there have been women veterinarians in the United States for a good many years, they failed to receive any particular amount of newspaper publicity until recently.

While on the subject of publicity received by the profession, we might mention the Twelfth International Veterinary Congress, which received considerable attention not only in the newspapers but in other places as well. Then may be mentioned the exhibit of the American Veterinary Medical Association at A Century of Progress in Chicago. More than sixteen million people passed through the gates while the 1934 Exposition was in progress, and records indicate that 70 per cent of those who visited the grounds entered the Hall of Science, in which the A. V. M. A. exhibit was located. This means that more than eleven million people had an opportunity to see the exhibit this year.

Reference has been made elsewhere to guidance leaflet 18, entitled "Veterinary Medicine," published by the Office of Education of the United States Department of the Interior several years ago. This leaflet has been given very wide distribution and it has been read by hundreds, probably thousands, of high school students at a time when they were trying to decide what they should take up for a career. Many letters have been received from these young people, asking for further information concerning the possibilities of the veterinary profession. Several other

publications are available, designed to be of assistance to persons who are about to select careers, and in every one of these that has come to our attention the veterinary profession has been presented in a very favorable light. In at least one instance, it is pointed out that, of all the professions, veterinary medicine probably is the only one that is not crowded.

Figures are available to show that about one out of every eight veterinary students now in college, is either the son, daughter or nephew of a veterinarian. This means only one thing and that is that many veterinarians have no hesitation in recommending their children or nephews to study veterinary medicine.

Letters received from the deans of the various colleges leave no doubt of the desirability, if not the necessity, of placing some restrictions on the number of students entering our veterinary colleges in the very near future. If there is anything we do not want it is an oversupply of veterinarians and, until the present year, there has not been anything to indicate that such an eventuality might happen. Every one knows that every student who presents himself for matriculation in a veterinary college will not make a good veterinarian. With the increased interest in veterinary medicine, shown by the large number of new students this year, a wonderful opportunity is presented for putting into practice what might be termed a system of selective admissions. As a matter of fact, such a plan is already in operation in at least two institutions. At the same time, another very strong argument is presented for the so-called five-year curriculum in veterinary medicine. From the facts at hand, there can no longer be any reasonable fear that adding a year to the course will materially reduce the number of students seeking admission to our veterinary colleges.

Every college in the list shows an increased enrollment over last year, with the exception of Cornell University. This statement can be made with credit to the Ithaca institution, for the reason that Dean Hagan reports that a total of 116 complete applications were received for admission to the freshman class this year, but 80 of these were declined. For the first time, the New York State Veterinary College this year was given the privilege of restricting admissions, and in this instance, the number of students finally accepted was reduced, by a process of selective admissions, to approximately one-third of those who had sought enrollment in the veterinary course. The number of freshmen actually matriculated was 36. Similar situations confronted the authorities in a number of the other institutions, and these situations were met in a variety of ways.

Dean Brumley, of Ohio State University, reports that the enrollment in the College of Veterinary Medicine this year is 278, an increase of 48 over one year ago. The number of veterinary students would have been much higher if it had not been for the fact that the matriculation of new students practically was stopped after August 15. Dean Brumley goes on to say that there seems to have been an unprecedented interest in veterinary medicine this year, and it became absolutely necessary to curtail the enrollment of new students about the middle of August. Dean Brumley is of the opinion that the number of veterinary students may now be too large, and with this thought in mind, a program is being formulated to regulate admissions and to select a number of freshman students at the beginning of the next academic year that appears to be in keeping with the facilities of the institution and the demands for veterinarians in the territory served by Ohio State University.

Dean Dick, of the University of Pennsylvania, in reporting an enrollment of 58 freshmen this year, stated that this number is larger than the School of Veterinary Medicine can accommodate, but that this condition could not be helped. Despite a similar experience a year ago, Dean Dick found that his institution again was confronted with practically the same situation, although steps had been taken to avoid it. As a result, the number of veterinary students this year is larger than can be handled properly with the facilities at present available. However, it would appear, from a comparison of the figures this year with those for previous year, that a weeding process has been going on at Pennsylvania. Of an original freshman class of 47 that entered three years ago, only 24 are enrolled as seniors this year. Last year, 40 sophomores were reported, but the junior class this year numbers only 36. Last year, 55 freshmen were reported, but this year there are only 44 sophomores.

Dr. C. D. McGilvray, principal of the Ontario Veterinary College, states that the freshman class at Guelph this year is the largest since 1914 and that the class of 66 freshmen will tax the teaching facilities of the institution. Additions to the faculty have been made, and the laboratory and clinical accommodations have been rearranged. Dr. McGilvray indicated that it had been necessary to refuse a large number of prospective students from the United States, as it was felt that the institution would be taxed even to take care of Canadian students.

Dean Stange, of Iowa State College, reports a total enrollment of 210 veterinary students this year as against 171 for last year. The number includes 64 pre-veterinary students and 62 registered

as freshmen. The sophomore, junior and senior classes at Ames are relatively small, the senior class of 14 being the smallest for a good many years. Dean Dykstra, of Kansas State College, reports a total enrollment of 230 students this year, an increase of 52 over one year ago. The figure given includes 59 pre-veterinary students. The number of senior students enrolled at Manhattan this year is greater than the combined number of sophomore and junior students.

Dean Giltner, of Michigan State College, reports 105 veterinary students enrolled this year, of whom 32 are freshmen, one of the largest first-year classes ever to be enrolled at East Lansing. The senior class of 30 is the largest in the history of the Michigan institution. Dean Newsom, of the Colorado Agricultural College, reports 120 students enrolled this year, an increase of eight over one year ago. In this number are 33 pre-veterinary students. The senior class at Fort Collins this year is one of the largest ever enrolled, with 35 scheduled to be graduated in 1935.

Dean Francis, of Texas A. & M. College, reports a freshman class of 54, the largest in the history of the institution. As a matter of fact, the total enrollment of veterinary students at College Station a year ago was only 61. Dean Wegner, of the State College of Washington, reports 65 freshmen enrolled. It is not necessary to go back very many years to find the time when

Veterinary student enrollment for the college year 1934-1935

	Fr.	Soph.	Jun.	Sen.	Spec.	Grd.	Tot.	1933–34	Chg.
Alabama Poly. Inst.	35	21	22	20	17	0	115	94	+21
Colorado Agr. Coll.	21	12	19	35	33*	0	120	112	+ 8
Cornell Univ.	36	34	29	58	0	6	163	175	−12
Iowa State Coll.	62	30	27	14	76†	1	210	171	+39
Kansas State Coll.	50	25	21	58	75‡	1	230	178	+52
Michigan Sta. Coll.	32	19	20	30	0	4	105	87	+18
Montreal, Univ. of.	15	8	6	10	0	0	39	36	+ 3
Ohio State Univ.	78	18	55	54	66*	7	278	230	+48
Ontario Vet. Coll.	66	33	46	38	0	1	184	151	+33
Penna., Univ. of.	58	44	36	26	6	0	170	165	+ 5
Texas A. & M. Coll.	54	30	13	13	3	1	114	61	+53
Washington, S. C. of	65	33	15	24	0	1	138	90	+48
Totals (1934–35)	572	307	309	380	276	22	1866	1550	+316
Last year	331	338	387	314	147	33	1550	1507§	+43

*Pre-veterinary students.
†Includes 64 pre-veterinary students.
‡Includes 59 pre-veterinary students.
§Total number of students for 1932-33.

this number was greater than the total number of students registered in all four classes.

Dean Cary, of Alabama Polytechnic Institute, reports a total of 115 veterinary students, the largest number ever enrolled at Auburn. The University of Montreal reports 39 registered in the veterinary school at Oka, Quebec, a modest increase of three over last year.

Detailed figures on the enrollment are given in the accompanying table.

DOCTOR EICHHORN RETURNS FROM EUROPE

A letter has just been received from Dr. Adolph Eichhorn, upon his return to New York from a trip abroad. Shortly after the close of the Twelfth International Veterinary Congress, Dr. Eichhorn felt the need of a rest following his strenuous labors of the past summer and decided to take a trip to Europe. He visited Budapest and, while there, saw Prof. Hutyra, who was very much pleased to have a first-hand report from Dr. Eichhorn on the transactions of the Congress. While in Berlin, a dinner was tendered Dr. Eichhorn, and many of those who attended the Congress in New York were present. Our German colleagues were quite lavish in their expressions concerning the Congress, particularly with regard to the social side. Dr. Eichhorn reports that, while in England, he heard nothing but praise for the arrangements and the results of the Congress.

BE CAREFUL WHAT YOU SIGN

Following the warning concerning collection agencies, published in the JOURNAL last month, additional information has been received, to the effect that veterinarians are being solicited to turn over their slow accounts to representatives of an agency that takes just about 100 per cent of any collections they are able to make. An investigation of the methods used by one of these concerns is being made, and a copy of the contract which veterinarians are being asked to sign has been secured and turned over to the legal counsel of the A. V. M. A. The provisions of this contract are such that it practically amounts to a fraud upon any one who signs it. Until the present investigation is completed, veterinarians are warned against entering into this type of contract with any collection agency with which they are not personally acquainted. More later.

APPLICATIONS FOR MEMBERSHIP

(See July, 1934 JOURNAL)

FIRST LISTING

JOHNSON, SAMUEL A. Broad St., Kinderhook, N. Y.
D. V. M., Cornell University, 1929
Vouchers: Harold C. Parker and J. G. Wills.

PAGE, H. F. Odon, Ind.
D. V. M., Ohio State University, 1932
Vouchers: J. L. Kixmiller and H. Busman.

PERSON, O. H. Wahoo, Nebr.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: Dan W. Hurst and Frank Breed.

PETERSON, W. C. Sleepy Eye, Minn.
D. V. M., Kansas City Veterinary College, 1915
Vouchers: W. A. Anderson and C. W. Olson.

SKILLEN, DONALD R. 751 Foothill Blvd., La Canada, Calif.
V. M. D., University of Pennsylvania, 1932
Vouchers: G. W. Closson and W. L. Curtis.

STEVENSON, J. C. 290 Post Ave., Westbury, N. Y.
D. V. M., Cornell University, 1932
Vouchers: J. Stuart Crawford and Harry P. Aronson.

WARWICK, B. L. Texas Agricultural Experiment Station, College Station, Texas
D. V. M., Iowa State College, 1919
M. S., University of Wisconsin, 1922
Ph.D., University of Wisconsin, 1925
Vouchers: F. B. Hadley and R. P. Marsteller.

Application Pending

(See October, 1934 JOURNAL)

SECOND LISTING

Robertson, Alex, Singapore Dairy Farms, Ltd., Bukit Panjang, Singapore, Straits Settlements.

The amount which should accompany an application filed this month is $5.83, which covers membership fee and dues to January 1, 1935, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

New Hampshire Veterinary Medical Association. State Capitol Building, Concord, N. H. November 2, 1934. Dr. F. S. Gray, Secretary, 8 Rogers St., Plymouth, N. H.

Central New York Veterinary Medical Association. Onondaga Hotel, Syracuse, N. Y. November 6, 1934. Dr. W. B. Switzer, Secretary, R. 5, Oswego, N. Y.

Connecticut Veterinary Medical Association. Hotel Garde, New Haven, Conn. November 7, 1934. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. November 7, 1934. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. November 7, 1934. Dr. Harley B. Wood, Secretary, 2754 Meramec St., Saint Louis, Mo.

Michigan-Ohio Veterinary Medical Association. Adrian, Mich. November 8, 1934. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Tulsa County Veterinary Association. Tulsa, Okla. November 8, 1934. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Interstate Veterinary Medical Association. Elks Building, Omaha, Nebr. November 12, 1934. Dr. G. L. Taylor, Secretary, Plattsmouth, Nebr.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. November 13, 1934. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

San Diego County Veterinary Medical Association. San Diego, Calif. November 13, 1934. Dr. L. K. Knighton, Secretary, 3438 Mountain View, San Diego, Calif.

Hudson Valley Veterinary Medical Society. Poughkeepsie, N. Y. November 14, 1934. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. November 14, 1934. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. November 20, 1934. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. November 21, 1934. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

Keystone Veterinary Medical Association. Philadelphia, Pa. November 28, 1934. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

National Association of B. A. I. Veterinarians. Hotel La Salle, Chicago, Ill. December 5-7, 1934. Dr. F. A. Imler, Secretary, Box 187, Kansas City, Kan.

United States Live Stock Sanitary Association. Hotel La Salle, Chicago, Ill. December 5-7, 1934. Dr. O. E. Dyson, Secretary, Live Stock Exchange Building, Wichita, Kan.

East Tennessee Veterinary Medical Society. White Surgical Supply Building, Knoxville, Tenn. December 8, 1934. Dr. R. E. Baker, Secretary, Morristown, Tenn.

Nebraska State Veterinary Medical Association. Omaha, Nebr. December 11-12, 1934. Dr. E. C. Jones, Secretary, c/o Platte Valley Serum Co., Grand Island, Nebr.

Western New York Veterinary Medical Association. Buffalo, N. Y. December 13, 1934. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

American Association for the Advancement of Science. Pittsburgh, Pa. December 27, 1934-January 2, 1935. Dr. Henry B. Ward, Secretary, Smithsonian Institution Building, Washington, D. C.

Minnesota State Veterinary Medical Society. Hotel Lowry, Saint Paul, Minn. January 3-4, 1935. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 8-9, 1935. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 10-11, 1935. D. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 14-15, 1935. Dr. C. H. Fauks, 1719 S. W. 15th St., Oklahoma City, Okla.

Ohio State Veterinary Medical Association. Columbus, Ohio. January 16-17, 1935. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 21-25, 1935. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Iowa Veterinary Medical Association. Fort Des Moines Hotel, Des Moines, Iowa. January 22-24, 1935. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

SERUM CONCENTRATE-LIVING VIRUS IMMUNITY AGAINST CANINE DISTEMPER*

Second Report

By GEORGE WATSON LITTLE, *New York, N. Y.*

Since the publication of our preliminary report[1] about a year ago, every effort has been made to substantiate the claims made at that time for the efficiency of the serum concentrate-living virus method of prevention and the serum concentrate treatment of canine distemper. The publication of further reports has purposely been withheld until such proof could be obtained by competent authorities who have carefully checked the original findings. It is fortunate that in all biological work there is a strict but unwritten law that one must prove unequivocally every statement that is made. It would not be sufficient to state that one has found this or that to be true, but in order to substantiate the statements in fact, many other competent authorities are given an opportunity to check and tear down the structural proof. After twenty-six months of intensive research and study in canine distemper, the author still has an open mind on the subject, although convinced regarding certain ideas tentatively advanced in the preliminary report. In the event of any new developments, a supplementary report will be forthcoming.

Quoting from the preliminary report and for the benefit of those who are not familiar with this work, it is sufficient to state that a single simultaneous injection of concentrated serum and living virus has been found to produce a strong and lasting immunity against canine distemper. Efforts have been made to simplify the simultaneous injections by the optional administration of the serum concentrate subcutaneously, intravenously or intraperitoneally, and of the virus subcutaneously instead of intradermally. Veterinarians engaged in the daily routine of practice can appreciate that the technic of administration should be made as simple as possible.

In the preliminary report was pointed out what was then considered the necessary precaution of first giving serum concentrate alone in doubtful or exposed cases. As shown in chart 1, it has been found that it is not so important to give a preliminary dose of serum concentrate. If one suspects that the animal may be incubating distemper as a result of previous exposure, it

*Presented at the first semi-annual meeting of the American Animal Hospital Association, New York, N. Y., August 17, 1934.

is now suggested that these cases be given the simultaneous treatment, kept under observation and treated with more serum concentrate, if necessary, rather than to withhold the living virus on the first inoculation. The administration of the living virus with the serum concentrate does not inhibit the protective action of the latter or tend to hasten the breakdown later of what might prove to be a previously exposed case. This is an analogous situation to that existing in prophylactic vaccination against hog cholera by the serum-virus, or simultaneous, method.

Chart 1 contained detailed records of 20 cases (name and address of owner, telephone number, date, breed, age, sex, markings, history, examination for parasites, hospitalization record, treatment and observations at 3-month intervals) showing the abandonment of the preliminary injection of serum concentrate early in the investigation and the successful application of the serum concentrate-living virus single injection method.

From these records and the findings of other veterinarians, we do not hesitate to advise the administration of serum concentrate and living virus simultaneously, notwithstanding that the patient is known to have been exposed to distemper before the injection. It was found that when these cases of previous exposure developed distemper, the disease was of a mild form which could be controlled readily by administering more serum concentrate alone, as shown in chart 2. This class of exposed patients does not constitute much of a problem if they are treated in the above manner.

Chart 2 contained detailed records of 20 dogs believed to have been exposed to distemper, which had received the single simultaneous injection of 3 cc of serum concentrate and living virus. Three of the dogs developed distemper in a mild form which was easily controlled by the subsequent injection of one 3-cc dose of serum concentrate alone.

Veterinarians in the metropolitan districts, especially in this country, are faced with the problem of immunizing dogs where the question of previous exposure is probable or at least doubtful. Puppies procured at pet shops, humane society shelters, dog shows, by shipping or otherwise exposed, constitute a large percentage of those presented for vaccination. It was therefore recognized in the beginning that some agent should be employed which would provide immediate protection for the animal so that attempts actually to immunize possibly or probably exposed patients could be carried out. It was known that a vaccine contains no antibodies and that it requires from one to two weeks for protection to result from this agent. Likewise, it was known that serum administered simultaneously with vaccine so modified

its antigenic properties for stimulating the formation of antibodies that a number of subsequent doses of vaccine were necessary. This, of course, is not a practical procedure from the standpoint of the owner or the veterinarian. In other words, we must have an agent which will meet and satisfy the exigencies of routine practice.

As shown in the preliminary report, we were convinced that serum concentrate possessed such marked advantages over unconcentrated serum, that it offered greater possibilities for both prevention and treatment. The freedom from inert protein fractions, the smaller dose and the intense concentration of antibodies not only eliminated undesirable systemic and local reactions but also insured quicker and more thorough absorption. With this in mind, we began to immunize young puppies ranging from seven weeks to three months of age, to determine what reaction, if any, would occur. It was soon found that no reaction occurred in these young puppies and, as shown in chart 3, they were strongly resistant to subsequent exposure.

Chart 3 contained detailed records of 20 puppies, seven weeks to three months of age, which were immunized by the simultaneous injection of serum concentrate and living virus without showing any reaction. All of these puppies were strongly resistant to infection when subsequently given field exposure to distemper.

Going further, we continued to inject these young animals and to hospitalize them in the presence of patients sick with distemper. It was soon observed that dogs of any age could be hospitalized and placed in the same cage with clinical cases suffering with this disease. It is desired to emphasize the fact that throughout this investigation no facilities were at hand for the isolation of distempered animals, nor was any attempt made to exclude them from the main ward of the hospital. As most of the younger puppies were found, upon microscopic examination, to be infested with one or more varieties of worms, the ideal procedure appeared to be to vaccinate these puppies immediately upon presentation at the hospital and to treat them for worms at the same time. Tetrachlorethylene proportionate to body weight was administered to these patients immediately after vaccination and the usual saline irrigation employed several hours later. For purposes of experiment, these puppies then were placed in the same kennels with active clinical cases of distemper. Repeating this procedure on many such patients proved that dogs of all ages could be vaccinated and "wormed" at the same time. For many years we have performed the cecum operation for the removal of whip-worms. The next step appeared to

be to determine if a susceptible dog could be immunized one day and operated upon the next. We proceeded to do so and have never found any necessity for being cautious about such procedure. It has solved the problem of the safe immunization of susceptible dogs under the most trying conditions, yet under the usual conditions of everyday practice. If this agent is to be successful in its entirety, it must meet the conditions which prevail and over which the veterinarian has no control. It is believed that the serum concentrate-living virus method will do just this.

From the inception of this investigation, an accurate record has been kept of every case that we attempted to immunize. Original records, dating back to May 4, 1932, are available. At intervals of every three months, inquiry has been made to determine whether any of these animals ever developed distemper. It is possible, therefore, to present and refer to these records (charts 4, 5 and 6), citing cases here and there which will prove that dogs can be immunized, wormed, or operated upon, on successive days.

Chart 4 contained detailed records of 20 cases varying in age from seven weeks to two years, which indicated that puppies as young as seven weeks could be immunized against distemper and treated for worms on the same day or on successive days.

Charts 5 and 6 contained detailed records of 40 cases showing that puppies as young as seven weeks were successfully immunized against distemper and treated for worms or operated upon the same day, or successive days, if they had not been exposed previously to distemper. All 40 animals were subsequently given field or hospital exposure.

As 4 cc seemed to hold the living virus successfully, the next step seemed to be to determine whether 3 cc would be just as effective when applied to dogs which had *not been exposed* to distemper. It was found that this dose was just as safe, apparently, as the 4-cc dose, as shown in chart 7.

Chart 7 contained detailed records of nine puppies, showing that 3 cc of serum concentrate was just as effective as 4 cc in protecting animals which had not been exposed to distemper.

Next, our failures will be submitted, and the causes explained to the best of our ability. The next charts record a number of dogs immunized with a serum concentrate prepared by hyperimmunizing immune dogs by a well-known method, which was abandoned as being inefficient early in the investigation. It was necessary, however, to prove that this method of hyperimmunization did not produce a serum potent enough to hold the living virus. This was the purpose of the experiment, the results of which are shown in charts 8 and 9.

Chart 8 contained detailed records of 20 dogs, each injected with 3 cc of serum concentrate prepared according to a well-known method of hyperimmunization. Seven of the animals died of distemper, three developed symptoms of distemper but recovered, and ten were reported in good health ten to twelve weeks following immunization.

Chart 9 contained detailed records of ten dogs, all of which failed to be protected by a dose of 3 cc of serum concentrate prepared by a well-known method of hyperimmunization. Four of the dogs died of distemper and six recovered after developing slight attacks of the disease.

Now are submitted records of 35 puppies including a group of 19 cocker spaniels, an Irish setter and a dachshund, which were given the serum concentrate and 0.25 cc of a 20 per cent suspension of frozen virus prepared from a neighborhood strain. At the time, this virus was considered equal, if not superior, to any thing in potency that could be purchased. We have been forced to change our mind. These 21 dogs were vaccinated on January 11 and some of them were exhibited at the Madison Square Garden. Several weeks later, it was quite evident that as a result of one dog's contact with an active case of distemper he had brought the infection home to the kennels. One by one, the majority of these 21 dogs became ill, with gastro-enteritis symptoms predominating. By administering large and frequent doses of serum concentrate, we were able to save every animal, as shown in charts 10 and 11.

Chart 10 contained detailed records of 20 puppies, three to nine months of age, showing the unsatisfactory results which followed an insufficient virus reaction, which produced only a temporary immunity. The virus used was a 20 per cent suspension of a neighborhood strain. All recovered following treatment with serum concentrate.

Chart 11 contained detailed records of 15 puppies, twelve weeks to five months of age, which developed only a temporary immunity following an insufficient virus reaction. The virus used was a 20 per cent suspension of a neighborhood strain. All developed symptoms of distemper. Five were treated with serum concentrate, of which one died and four recovered. Ten were given medication without serum concentrate and all recovered.

The controversial aspects of this subject will be treated with brevity. It is well known and apparent that there are difficulties to overcome in the standardization of all antisera. One of our associates has been of great assistance in our research concerning the subject of standardization. Knowing that he had been working on the Schilling blood-picture, we called upon him for assistance in this investigation. He has brought out some very interesting points as to the characteristics of the blood of dogs: first, confirming the normal blood-count of these animals; and, second, studying intensively the characteristics and deviations

of the entire blood-picture in distemper.[2,3] It would appear that there is a ray of hope that a satisfactory method of standardization will be found through the work of Dr. Mark Loren Morris. He is to be most highly commended for the thoroughness of his study along these lines. His researches have not been brought to the stage of conclusion where we can announce that any definite method of standardization of anti-canine distemper serum is possible. It is a very difficult problem which, thus far, has defied our best efforts to solve.

In a recently published paper,[4] Dr. Morris shows a number of charts concerning his experiments which strongly indicate the efficacy of serum concentrate to protect against three different viruses used in his experiments. He was likewise successful in proving that 3 cc or even 2 cc of serum concentrate would protect against viruses of known virulence. The author also has shown by experiments that the simultaneous injection of the serum concentrate and living virus in puppies two to four months of age appears to produce a lasting immunity.

It is interesting to note that Dr. Morris subjected his vaccinated dogs to immediate exposure by placing them in the distemper ward in contact with active cases of the disease. Moreover, one dog remained there for 130 days. His efforts to break down the immunity by injecting, three times a week, a fresh spleen virus of known virulence failed to produce any reaction. This work appears to confirm our findings along these lines.

A careful study of the blood-picture of the animals which were studied by Dr. Morris points out that a marked reaction appears in each instance between the tenth and 16th days. This reaction consists in the lowering of the total white corpuscle count, an increase in the number of immature leucocytes and a lowering of the nuclear index. In comparing these pictures with a virus picture, a close similarity was noted. It would seem, therefore, that the dog actually had been attacked by the virus, the development of which had been controlled by the serum concentrate. The above is an almost exact quotation from Dr. Morris' report.

The question of the filtrable virus will be referred to very briefly. We are content to rest our case almost entirely on the findings of Laidlaw and Dunkin,[5-8] whose researches on this subject have been checked and verified by many outstanding authorities.

We are not especially interested in discussing methods of hyperimmunization of immune dogs for the purpose of producing homologous serum against distemper. Reference is made only to

what we have attempted to prove in the records submitted here. Our method is not unlike those in commercial use today, other than that all immune dogs intended to be used as serum-producers are hyperimmunized over a much longer period of time than what we believe to be generally the case.

With reference to the concentration of the serum concentrate, the method that we have used is not unlike those already in commercial use today.

Early in this investigation, it was realized that the best method of proving the efficacy of the serum-virus treatment was to apply it directly in the field of everyday practice. Certain hypotheses were proved by the author, but these required verification by other workers. The method of prosecuting these investigations differed from the usual methods in that we felt it necessary to have extensive tests conducted under actual field conditions rather than to draw our conclusions from laboratory or technical proof entirely. It was fortunate, therefore, that the decision was made to distribute the serum concentrate to prominent practicing veterinarians in different parts of the United States, Canada and Australia. The reports which will be submitted here confirm the findings of the author. The report of Dr. F. T. Wheatland, Assistant Director of the Department of Health, Commonwealth of Australia, proves that the serum concentrate protects against the Australian virus when injected simultaneously and also when the virus is administered 72 hours prior to the serum concentrate.

We are thoroughly convinced that the simultaneous injection of serum concentrate and living virus, as compared with the "pre-virus" method of immunization, is well adapted for use under clinical and hospital conditions. It establishes a degree of immunity which affords a strong protection against distemper; moreover, it is much more practical in its adaptation to everyday practice. We have pointed out, however, that a stronger and more lasting immunity can be established if the animals so injected are immediately exposed to the disease, even to the extent of placing them in the distemper ward in the presence of active cases of distemper. Our own records and those of our associates offer proof of the above statements.

The pre-virus-serum concentrate method, as compared with the simultaneous method, is much better adapted to the prevention of distemper in large kennels, especially of the more susceptible sporting breeds.

Since the publication of the preliminary report, our investigations indicate that a stronger immunity develops, due to a greater

virus reaction, when the virus is injected alone 48 to 72 hours before the serum concentrate is given. This finding is in keeping with that recently reported by the English investigator, Boddie.[9] (See tables I, II, III and IV.)

TABLE I—*Results of experiment to determine whether concentrated homologous anti-canine distemper serum in doses of from 2 to 5 cc will protect against a 20-mg dose of virus administered 48 hours before the serum.*

Dog	Breed	Weight (Lbs.)	Temperature Before Treatment	Serum	
				No.	Dose (cc)
6541	Hound	12	101.2	359A	2
6542	Hound	10	102.0	354	2
6543	Hound	10	101.6	359A	3
6544	Hound	10	101.4	354	3
6545	Hound	12	102.0	359A	4
6570	Mongrel	12	102.0	354	4
6571	Mongrel	14	101.8	359A	5
6572	Mongrel	17	101.2	354	5
6573	Mongrel	15	101.4	Control	

Note: Dogs in this experiment were three months old and in good condition. Virus (Exp. 363) administered, May 25, 1934, and serum 48 hours later. These dogs continued in good physical condition, with the exception of the control (6573), which showed visible symptoms of distemper during the fourth week. On June 28, 1934, all were given 0.25 cc of fresh 20 per cent spleen virus. One week after this inoculation exposure, elevations of temperature were observed. However, the dogs continued in good physical condition to the end of the observation period, July 19, 1934. The control developed marked symptoms, including emaciation, and was destroyed, July 19, 1934. See table II for record of daily temperatures.

By way of summarizing this pre-virus-serum injection work, we have proved that the subcutaneous administration of the living virus, 48 to 72 hours previous to the injection of the serum concentrate, confers an even greater and more solid immunity than the simultaneous injection. It is advised, therefore, that when the exigencies of practice are such that animals can be hospitalized, the virus should be given in the above manner. It is obvious, however, that dogs could not be given the virus and then released from the custody of the veterinarian. Experience teaches us that, for reasons beyond our control and often beyond the control of the owner, a certain percentage of these patients might not be returned to have the immunization completed. It would, therefore, be a most unfortunate position in which the veterinarian would be placed, if in a single case he should be subjected to criticism for giving the virus alone and thereby be guilty of disseminating the disease, in the event that the owner, for some

unknown reason, should fail to return the dog for its injection of serum concentrate.

It is quite evident that the single simultaneous injection overcomes this hazard and for all purposes of everyday practice will be found to be the most popular method of treatment. We must

TABLE II—*Temperatures of nine dogs in table I.*

DATE (1934)	6541	6542	6543	6544	6545	6570	6571	6572	6573
5–25	1.2	2.0	1.6	1.4	2.0	2.0	1.8	1.2	1.4
5–26	0.6	0.4	1.2	0.4	1.0	1.6	2.6	2.4	1.6
5–27	0.4	1.0	1.4	2.0	0.0	1.8	2.0	2.0	2.0
5–28	2.0	1.8	2.2	2.0	2.0	3.2	2.8	2.8	2.0
5–29	1.2	1.0	1.0	1.4	1.4	2.0	3.0	2.2	2.4
5–30	1.0	1.2	1.4	1.6	1.2	1.2	2.4	2.4	4.0
5–31	2.0	2.0	1.8	1.6	1.0	1.0	3.8	2.8	3.6
6–1	1.6	1.4	2.0	1.6	1.0	2.2	2.4	2.0	2.6
6–2	1.4	5.0	1.8	1.0	1.6	3.0	2.6	2.4	2.4
6–3	1.8	1.8	1.0	1.6	0.0	0.4	2.0	2.4	2.2
6–4	1.8	2.0	1.4	1.8	1.6	3.2	4.4	4.0	3.4
6–5	1.4	2.6	2.0	0.0	1.0	1.8	2.8	5.0	3.0
6–6	1.0	0.8	1.2	1.2	1.0	3.0	1.4	2.4	3.0
6–7	2.0	1.2	1.6	1.8	2.0	2.0	1.0	1.8	2.6
6–8	0.8	1.2	1.4	0.0	0.6	1.0	3.0	4.0	3.0
6–9	1.0	1.4	1.2	1.6	1.4	2.0	1.6	2.6	1.4
6–10	1.0	0.8	1.8	1.0	1.6	1.4	2.4	2.0	3.0
6–11	1.6	1.0	1.0	1.0	0.6	1.0	1.0	0.8	1.0
6–12	0.6	1.0	1.8	1.0	1.2	1.0	2.6	2.0	3.2
6–13	1.0	1.6	1.8	1.0	1.8	1.6	1.8	2.8	2.8
6–14	1.0	0.8	1.0	1.0	0.8	1.0	2.0	2.0	1.8
6–15	1.6	2.0	2.0	2.8	2.6	1.2	2.8	2.4	2.0
6–16	2.4	1.4	1.6	1.0	1.4	3.4	4.0	3.0	3.4
6–17	1.0	1.6	1.2	1.2	1.0	3.0	3.0	2.8	2.0
6–18	1.8	0.8	1.0	1.4	1.6	2.0	2.0	2.4	1.0
6–19	1.8	1.4	1.6	1.0	2.0	1.8	1.0	1.6	2.0
6–20	0.8	1.2	1.8	2.0	1.0	2.4	2.0	1.8	1.0
6–21	1.2	1.0	1.6	1.4	1.0	1.0	1.0	1.0	1.0
6–22	0.8	0.8	1.0	1.0	1.8	2.0	2.4	2.0	1.0
6–23	1.4	1.4	1.2	1.6	1.6	1.6	1.6	1.8	0.4
6–24	1.0	1.0	1.6	1.0	2.8	1.2	2.2	2.0	1.2
6–25	2.2	1.6	1.8	2.0	2.4	2.2	2.8	2.2	2.4
6–26	0.8	1.6	1.2	1.4	1.0	2.0	4.0	2.2	1.0
6–27	1.2	1.4	1.4	2.4	1.4	2.0	2.0	1.8	1.0
6–28	2.0	2.0	1.8	2.4	2.0	1.8	2.0	3.0	1.6

emphasize that exposed patients, over which the veterinarian has no control, will always be a bone of contention and a sore spot which may defeat at any time the production of a solid immunity. Observation of these doubtful cases after the administration of the serum and virus is absolutely necessary, as the administration of more concentrated serum will always preclude the possibilities of any disastrous result.

The pre-virus-serum concentrate method produces a stronger immunity because it produces a stronger virus reaction in the animal itself. As the incubation period of distemper is from four to six days, there is no danger at any time of producing the disease by the pre-virus-serum injection if given according to recommendation. The serum concentrate, due to its high antibody content, is potent enough to neutralize this virus reaction so that there never is any danger of producing the disease.

TABLE III—*Results of experiment to determine whether the administration of concentrated homologous anti-canine distemper serum (Little) 72 hours after the injection of virus is a safe and effective immunizing procedure.*

Dog	Breed	Weight (Lbs.)	Temperature Before Treatment	Treatment	
				Virus	Serum
6053	Mongrel	5	101.8		
6054	Mongrel	8	102.0		
6055	Mongrel	6	101.6		
6056	Mongrel	5	101.6		
6057	Mongrel	5	101.8		
6058	Mongrel	7	102.4		4.0 cc
6058	Collie	12	102.0	10.0 mg	serum
6060	Collie	11	102.4	virus 006	Exp. 330
6061	Collie	12	102.2	March 28,	March 31,
6062	Collie	10	102.4	1934	1934
6063	Collie	8	101.4		
6064	Collie	11	102.4		
6065	Collie	11	102.4		
6066	Collie	10	101.2		

Note: Dogs in this experiment were two months old and in good condition. These 14 dogs remained in good physical condition throughout the five-week period of observation, from March 28 to May 1, 1934. On the latter date, these dogs were exposed to active cases of distemper. All remained in good physical condition until the end of the observation period, May 30, 1934, except dog 6057 which went off feed May 23 and died May 26. The cause of death was not determined, as the autopsy and bacteriological examination were negative. See table IV for record of daily temperatures.

It is apparent, therefore, in the light of the great mass of evidence obtained, that the pre-virus injection is superior to the simultaneous injection in all cases where it is practicable to carry it out. We realize that veterinarians will accept this with reservations in their own minds, being afraid of a possible virus reaction too strong to control. We can only reiterate that there is no danger.

THERAPEUTIC TREATMENT

Undoubtedly many here are as much interested in the treatment of distemper as in its prevention. We are aware also that

TABLE IV—*Temperatures of 14 dogs in table III.*

Date (1934)	6055	6054	6055	6056	6057	6058	6059	6060	6061	6062	6063	6064	6065	6066
3-28	1.6	1.4	2.0	2.2	1.8	1.4	2.0	1.6	0.0	1.8	0.0	0.2	2.0	1.2
3-29	1.2	1.8	2.0	2.2	2.0	2.0	2.0	2.4	2.8	2.2	2.4	2.4	2.4	2.6
3-30	2.8	3.0	2.0	1.8	1.8	2.0	3.4	2.0	2.0	1.8	2.0	2.2	1.8	2.0
3-31	3.0	3.0	3.2	2.6	1.8	2.2	2.0	2.0	2.4	2.6	2.4	2.2	2.0	2.8
4-1	1.4	2.0	1.8	2.0	1.6	1.8	2.4	2.4	2.2	2.2	2.0	2.2	2.4	2.0
4-2	1.8	1.8	1.6	1.8	1.6	1.8	2.0	1.8	2.0	1.8	1.8	2.0	1.6	1.6
4-3	1.6	2.2	1.8	2.0	2.0	2.2	2.4	2.0	1.8	2.0	1.6	2.0	2.4	2.4
4-4	1.8	1.8	2.0	1.8	1.6	1.4	1.6	1.6	2.0	1.6	1.2	1.8	2.0	1.4
4-5	2.0	1.4	2.0	2.0	2.2	2.4	2.2	2.0	2.0	2.0	1.2	2.4	2.6	2.0
4-6	1.6	2.0	2.0	2.2	2.0	2.6	1.4	2.6	2.4	1.8	2.4	2.0	2.4	1.2
4-7	1.6	1.4	1.6	2.0	1.4	1.6	2.0	1.8	2.0	1.6	1.6	2.4	2.2	2.2
4-8	1.8	2.2	2.6	2.0	2.0	2.4	2.4	2.4	2.8	2.0	2.6	2.6	2.8	2.4
4-9	2.0	2.4	2.0	2.2	2.0	2.4	1.8	2.0	2.4	2.6	1.0	2.6	3.0	2.2
4-10	1.6	2.0	1.8	1.6	1.8	2.0	2.0	2.0	2.2	1.8	2.0	2.0	2.2	2.0
4-11	2.0	1.6	1.6	1.8	1.0	1.6	2.4	2.4	2.4	2.2	2.0	2.4	2.8	2.6
4-12	1.6	1.8	1.0	1.6	1.6	1.0	1.0	2.2	1.8	1.4	2.0	2.0	2.6	1.8
4-13	1.8	1.6	2.0	1.2	1.2	2.2	1.4	1.6	1.0	1.4	2.0	2.0	1.8	1.2
4-14	1.6	2.8	2.0	1.8	1.8	2.0	1.8	1.4	2.0	2.4	1.8	1.0	1.6	1.4
4-15	1.6	1.8	1.6	2.0	1.8	1.6	1.6	2.0	2.0	1.6	2.2	1.8	1.6	1.8
4-16	1.0	1.8	2.0	1.8	2.0	1.2	2.0	1.4	2.0	1.6	1.8	2.4	2.0	1.8
4-17	1.4	2.2	1.8	1.0	1.4	2.4	1.0	1.8	2.0	1.8	1.8	2.4	2.2	1.6
4-18	1.8	2.0	2.6	2.4	1.4	2.4	1.8	2.0	2.2	3.2	1.4	2.2	2.4	1.8
4-19	0.4	1.0	1.0	0.4	0.4	1.0	1.8	1.2	1.6	1.8	1.0	1.4	2.0	1.2
4-20	1.8	2.6	2.2	2.6	2.0	2.4	2.6	2.4	2.4	2.4	2.2	2.2	2.8	2.0
4-21	2.0	2.0	1.8	1.8	2.2	1.8	1.8	1.6	1.8	1.8	2.0	1.8	2.2	2.0
4-22	1.6	1.8	2.0	1.0	1.4	1.6	1.4	1.0	1.6	1.8	1.8	2.0	2.4	2.2
4-23	2.0	2.6	2.8	2.6	1.4	2.0	1.6	1.4	1.8	1.6	2.0	2.2	1.8	1.8
4-24	1.6	2.0	2.2	2.4	1.2	1.8	1.8	1.6	2.2	2.0	1.4	1.8	1.4	2.8
4-25	1.8	2.6	2.0	2.8	2.4	2.2	2.0	2.0	1.8	2.8	1.6	1.6	2.6	3.4
4-26	2.0	2.2	2.4	2.8	1.8	2.2	2.0	3.4	1.6	2.4	1.2	1.8	2.4	2.0
4-27	1.8	1.8	1.4	1.6	1.4	1.0	3.4	2.8	1.0	2.0	1.0	1.6	1.4	2.0
4-28	2.6	1.6	1.6	1.8	1.0	0.6	1.6	0.4	2.0	1.6	1.6	1.0	1.4	1.4
4-29	2.2	1.4	1.3	2.4	2.6	1.6	1.6	1.6	2.4	2.4	1.4	1.8	2.8	2.6
4-30	1.6	2.0	1.8	1.6	1.8	2.0	1.4	1.2	2.0	2.0	1.0	2.0	1.6	2.2
5-1	1.8	2.0	1.6	1.6	1.8	1.0	3.0	2.4	2.2	2.4	1.4	2.4	2.4	1.2

the present curative agents which can be obtained have great room for improvement. We have searched for years for a truly efficient serum for the treatment of distemper. The only way that appeared practical was to increase in some way the potency of homologous serum. The evidence that has been submitted here seems to prove sufficiently that we are dealing with a highly potent product in the serum concentrate. Moreover, it is abundantly fortified with antibodies against the secondary invaders, as agglutination tests have shown. (See table V.)

This investigation was inspired by a desire to have a serum from which the inert proteins have been largely removed so that it may be used more readily for both curative and preventive purposes. This is the principal objection to unconcentrated homologous serum—its volume is too great for the amount of antibodies contained. Serum sickness, malaise and shock are most certainly factors and deterrents in the use of unconcentrated homologous serum for curative purposes. These adverse reactions can be attributed almost entirely to the excess of inert protein materials.

TABLE V—*Agglutination titres of homologous anti-canine distemper serum before and after concentration.*

Dog	Antigen	Dilutions and Reactions								
		100	200	500	1000	1500	2000	2500	3000	3500
2225	S	+	+	+	+	+	—	—	—	—
	B	+	+	+	+	+	—	—	—	—
	P	+	+	+	+	+	—	—	—	—
3421	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	+	p	—	—	—	—
2606	S	+	+	+	+	+	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	+	+	+	—	—	—
3569	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	+	+	—	—	—	—
3247	S	+	+	+	+	+	—	—	—	—
	B	+	+	+	+	—	—	—	—	—
	P	+	+	+	p	—	—	—	—	—
2255	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	+	+	+	+	p	—
3608	S	+	+	+	—	—	—	—	—	—
	B	+	+	+	+	+	—	—	—	—
	P	+	+	+	+	+	p	—	—	—
3396	S	+	+	+	—	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3054	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3610	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
2548	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	+	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
2054	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3561	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	+	+	+	+	p	—
3559	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3525	S	+	+	+	+	p	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	+	—	—	—	—	—
3451	S	+	+	+	—	—	—	—	—	—
	B	+	+	+	+	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3520	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	p	—	—	—	—	—

Dog	Antigen	Dilutions and Reactions								
		100	200	500	1000	1500	2000	2500	3000	3500
3521	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	+	—	—	—	—	—
	P	+	+	+	+	—	—	—	—	—
3819	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	p	—	—	—	—	—
3240	S	+	+	+	—	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	+	p	—	—	—	—
3255	S	+	+	+	+	+	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	+	—	—	—	—	—
3389	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	+	+	—	—	—	—
	P	+	+	+	+	—	—	—	—	—
3564	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	+	—	—	—	—	—
	P	+	+	+	+	+	p	—	—	—
3636	S	+	+	+	+	p	—	—	—	—
	B	+	+	+	+	+	—	—	—	—
	P	+	+	+	+	—	—	—	—	—
3250	S	+	+	p	—	—	—	—	—	—
	B	+	+	—	—	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3192	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	+	+	+	+	p	—
3566	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	p	—	—	—	—	—
2511	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
3243	S	+	+	+	+	+	—	—	—	—
	B	+	+	+	+	+	—	—	—	—
	P	+	+	+	+	—	—	—	—	—
3577	S	+	+	+	+	p	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	p	—	—	—	—	—
3161	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	+	+	—	—	—	—
2224	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	+	p	—	—	—	—
	P	+	+	+	+	+	+	+	—	—
3543	S	+	+	+	p	—	—	—	—	—
	B	+	+	+	—	—	—	—	—	—
	P	+	+	+	—	—	—	—	—	—
C.S.	S	+	+	+	+	—	—	—	—	—
	B	+	+	+	p	—	—	—	—	—
	P	+	+	+	+	+	+	+	p	—

Key: + = Complete agglutination. p = Partial agglutination. — = No agglutination. S = Streptococci (hemolytic and non-hemolytic). B = *Alcaligenes bronchisepticus*. P = Paratyphoid B (*Salmonella schottmülleri*) and *Salmonella enteritidis* (equal parts).

Note: Agglutination tests made with individual and composite samples before concentration and with one composite sample after concentration. The composite sample after concentration was positive for streptococci (hemolytic and non-hemolytic) in a dilution of 1:4,000; for *Alcaligenes bronchisepticus* in a dilution of 1:2,500; and for *Salmonella schottmülleri* and *Salmonella enteritidis* in a dilution of 1:10,000.

There is another advantage which is most important and should not be overlooked. In the removal of the inert proteins, we not only retain all of the antibody fractions, but render them more easily available for absorption by the patient. This is manifested by the sudden drop of temperature without shock in many dogs treated. When this temperature goes down rapidly in such a short time, it has a tendency to remain normal. On the contrary, it has been found that this is not true of unconcentrated homologous serum. Nature's efforts to eliminate a large amount of inert protein material tend to lower the resistance, so that in 48 hours the temperature of the dog rises again and more serum must be administered.

In view of the tremendous amount of homologous, unconcentrated serum that some of our veterinarians are giving as an initial dose, it renders this hypothesis quite obvious. From our own experience we find that there is no economy in giving a small initial dose. Many of our men are giving 60 to 100 cc of homologous serum at one dose at the beginning of an attack of distemper. The equivalent of this amount in serum concentrate is 12 to 20 cc. (See table I.)

Unquestionably one large dose of serum concentrate is of greater value than repeated small doses. Dogs in the incipient stages of distemper, uncomplicated with pneumonia or enteritis, will respond spectacularly to a large dose of serum concentrate. It is ideal, therefore, to inject such a dose equivalent to one-fifth the amount of unconcentrated serum, but so compact in volume that it does not suggest an overdose in the presence of an owner of a toy dog.

There is another school of veterinarians who favor smaller doses of serum in the treatment of distemper. They argue that the larger percentage of patients which they are called upon to treat have developed the disease to the point where the secondary invaders have obtained a foothold. It is beyond question that this is true, and more common in some parts of the country than others, due to the negligence on the part of owners of dogs to seek the services of the veterinarian until the animals are about to die. For this class of cases I can only suggest that there may be little advantage in giving a tremendous dose of serum in the beginning. Small, repeated doses are more applicable to those patients which we can class as no longer being true distemper cases but complicated ones, victims of the secondary invading organisms of the disease. For these patients, a relatively small dose of serum concentrate (4 to 8 cc), repeated every 24 to 48

hours, is advisable until the temperature becomes normal and remains so for 48 hours. We might class these patients as those in which we realize that we have a fight on our hands right from the beginning in order to save them. The use of biologics alone, and ignoring diet, chest inunctions and medicinal treatment, is not advised, for in the light of experience, the combination of the above appears to be the most sensible procedure.

Acknowledgments

Almost from the inception of this investigation, Dr. William J. Lentz, director of the Small Animal Clinic of the University of Pennsylvania, has coöperated with the author. His advice and counsel have been given freely and have been a source of great satisfaction and benefit. His patience in checking our experimental work is to be highly commended.

Acknowledgment is made of the great assistance given by our other associates: Drs. Abraham J. Klein, Victor Ross, and Mark Loren Morris. They have been of great help in the work.

Wonderful coöperation has been received from many prominent veterinarians throughout the United States and other countries. Their reports have been carefully prepared and have lent to this work an element of proof which could not possibly have been obtained in any other way.

Summary

1. A single, simultaneous injection of serum concentrate produces a strong and lasting immunity against distemper.

2. The intradermal injection of virus has no advantages over the subcutaneous injection, as pertaining to immunization by the serum concentrate-living virus method.

3. The serum concentrate-living virus method can be carried out regardless of exposure, with safety and effect on young puppies less than three months old, and coincident with "worming" or operative procedure.

4. In previously exposed cases, symptoms of distemper are controlled easily by a small additional dose of serum concentrate.

5. Usual methods of hyperimmunization do not produce a serum concentrate of sufficient potency to protect against living virus.

6. Frozen virus (20 per cent suspension) of a neighborhood strain failed to develop an immunity against distemper.

7. Records of field tests in the United States, Canada and Australia confirm the efficacy of the serum concentrate-living virus method of immunization.

8. A more lasting immunity is produced by *immediate exposure* after the single, simultaneous injection of serum concentrate-living virus.

9. The pre-virus-serum concentrate method of immunization, in which the virus is administered 48 to 72 hours previous to the serum concentrate, produces the strongest and most lasting immunity against distemper.

10. Serum concentrate as a curative eliminates unfavorable reactions, so frequently experienced with unconcentrated, homologous serum, when used in quantity.

11. Spectacular results have been obtained in the use of serum concentrate as a curative in true distemper during the initial stages.

REFERENCES

[1]Little, G. W., and Lentz, W. J.: Production of immunity to canine distemper by serum concentrate-living virus, simultaneous injections. Jour. A. V. M. A., lxxxiii (1933), n. s. 37 (3), pp. 405-409.

[2]Morris, M. L.: Laboratory and clinical diagnosis of canine distemper. No. Amer. Vet., xiv (1933), 3, pp. 34-48.

[3]Morris, M. L.: Further studies in the control and hospitalization of canine distemper. Jour. A. V. M. A., lxxxv (1934), n. s. 38 (1), pp. 39-63.

[4]Morris, M. L.: Clinical and laboratory studies of the simultaneous use of serum concentrate (Little) and living virus for immunizing dogs against distemper. No. Amer. Vet., xv (1934), 8, pp. 32-37.

[5]Dunkin, G. W., and Laidlaw, P. P.: Studies on dog distemper. I. Dog distemper in the ferret. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 201.

[6]Dunkin, G. W., and Laidlaw, P. P.: Studies on dog distemper. II. Experimental distemper in the dog. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 213.

[7]Laidlaw, P. P., and Dunkin, G. W.: Studies on dog distemper. III. The nature of the virus. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 222.

[8]Dunkin, G. W., and Laidlaw, P. P.: Some further observations on dog distemper. I. The durability of the immunity following vaccine and virus administration. Jour. A. V. M. A., lxxviii (1931), n. s. 31 (4), pp. 545-551.

[9]Boddie, G. F.: The clinical aspect of distemper immunization. Vet. Rec., n. s. xiv (1934), 19, pp. 507-510.

FURTHER OBSERVATIONS ON THE USE OF SERUM CONCENTRATE (LITTLE) TO CONTROL CANINE DISTEMPER*

By WM. J. LENTZ, *Philadelphia, Pa.*
Director of the Small Animal Clinic
Veterinary School, University of Pennsylvania

We are all agreed, I am sure, that canine distemper is still a vexing problem. Its causal factor has been demonstrated by Dunkin and Laidlaw unquestionably to be a filtrable virus, thereby confirming the findings of Carré. We can admit of no justification for controversy on that point in view of the findings of other

*Presented at the first semi-annual meeting of the American Animal Hospital Association, New York, N. Y., August 17, 1934.

investigators all over the world. This virus has been found to be the true cause of the disease whether it be at the equator or near the Arctic Circle, in the Orient, India, Continental Europe, the Americas or Australia. In fact, where dogs are found, this disease runs true to form, as far as its incitant factor is concerned.

Unlike some of the filtrable viruses, that of canine distemper is found to be so closely linked to the cellular structures of the spleen and lymph-glands and possibly other organs that we can call it truly intracellular in its nature. Dr. and Mrs. Wharton[1] have demonstrated this fact conclusively, it seems. It is true that during the initial rise of temperature, after incubation of the disease, we find and we are able to demonstrate the presence of the virus in the blood-stream. The blood at this period is said to be highly virulent, so that susceptible animals, injected with even a small fraction of one cubic centimeter, will react and develop a definite syndrome of distemper. For many years, it was evidently assumed that the filtrable virus was a free agent floating in the blood-stream, coming from nowhere in particular. Now we know that is not the case. The spleen appears to be the largest and most efficient factory for the production of the virus, with smaller factories located in the lymph-glands and possibly other organs of the animal body. If it were not for the intracellular character of the filtrable virus, our work with this disease would be greatly simplified. An abundance of this poison could then be harvested without recourse to our present methods of obtaining it from the spleen and lymph-glands. The question of its viability over a longer period would be simplified and possibly the weakness of its virulence, at times, could be overcome by purification. In spite of these handicaps, going back to the intracellular structure, the question of the virility and the viability of the filtrable virus has been sucessfully overcome so that a uniform potency can be assured at all times.

If it had not been for the apparently high potency of the serum concentrate, which was made manifest repeatedly in our experiments with it, I doubt that I ever would have consented to become associated with this work. In this case, it consisted of determining whether 4 cc of the serum would resist the virus, so as not only to prevent the production of the disease but also to confer a solid immunity. I have found, in a few cases, that apparently 3 cc or even 2 cc of the serum concentrate seems to "hold" the virus. Other investigators have checked Dr. Little's work and I believe they have found that this comparatively small amount of serum concentrate will control the virus. It is quite necessary, then,

to carry further these occasional tests of the serum concentrate and make it a routine practice, that every batch of serum, before it leaves the laboratory, be tested as to its antibody strength, estimated by its ability to "hold" the living virus by a safe margin. I believe that this is the present procedure and the future intention of those connected with its production.

It is quite evident that the solid immunity produced by the serum concentrate depends not only on its high antibody strength but upon the virus reaction.

In the treatment of distemper, I was a bit skeptical at first that large doses of serum had any more effect than moderately small and repeated doses. I am forced to believe that it is true that the initial dose of the serum concentrate should be sufficient to afford a tremendous stimulus in order to turn the tide toward recovery. When the secondary invaders have obtained a foothold, this is not true. In many instances I have found that the one injection of the serum is sufficient; other patients have required daily or 48-hour injections for several times, usually of a dose of about 4 cc. In my experience, there is no economy in giving a small dose in the beginning.

The concentration of the serum would seem to be a distinct advantage, to my mind, for one is excluding a great volume of waste material in the form of inert proteins.

In conclusion, I would like to express my reaction to this entire investigation. My findings in a general way seem to substantiate the claims of Dr. Little. It is true, he has the advantage of being a practitioner familiar with the problems in the field but, without contact with the laboratory, he would have been lost. It would now seem that this method of immunization should prove itself to be of marked value, particularly to those of us engaged in hospital work.

REFERENCE

[1]Wharton, D. R. A., and Wharton, M. W.: Amer. Jour. Hyg., xix (1934), 1, pp. 189-216.

EXPERIMENTS TO PRODUCE IMMUNITY TO CANINE DISTEMPER IN FERRETS*

By VICTOR ROSS, *New York, N. Y.*

Department of Bacteriology, New York University

The extreme susceptibility of the ferret to the virus of canine distemper demonstrated by Dunkin and Laidlaw[1] in the course of

*Presented at the first semi-annual meeting of the American Animal Hospital Association, New York, N. Y., August 17, 1934.

their extensive study of this disease offered an opportunity to learn how this animal would react to a simultaneous injection of living virus and concentrated antiserum. Such an injection is employed by Little[2] in immunizing dogs and has been found by him to involve no risk. The same technic was employed also with four different samples of unconcentrated serum purchased in the usual way.

The present experiments, employing a "side to side" injection of living virus and serum, are a test of whether the serum "holds" the virus *and* whether ferrets so treated will survive exposure to the disease as it exists in control ferrets injected with living virus alone. We cannot distinguish these effects. If an animal has died, we cannot tell whether death resulted from a failure of the serum to resist the virus or from exposure to the diseased controls, since the latter were in intimate contact with the serum-treated ferrets. The object was rather to duplicate Little's method of immunizing dogs, which permits immediate and continued exposure to sick animals, rather than to determine the mode of action. A difference in procedure (the exception will be noted below) consisted in the fact that exposure would not begin, in the case of the ferrets, until controls became ill, whereas Little subjects his vaccinated dogs immediately to contact with dogs which may or may not be sick with distemper, no attempt being made to avoid contact with the disease. The test is doubtless a much more severe one for ferrets than for dogs, since the serum employed is from another animal and consequently does not remain long as a protecting agent. At the time when controls have become ill, such serum undoubtedly has been entirely eliminated by the treated ferrets. No striking beneficial results were, therefore, anticipated. Survival would appear to depend primarily on whether the injected living virus and serum immunize ferrets sufficiently so that the danger of exposure to ill animals is safely resisted. Dunkin and Laidlaw found that ferrets are not immunized by formalinized canine virus.

All injections of serum and virus were made subcutaneously on opposite sides of the body. Two controls injected with Lederle desiccated virus (10 mg) became ill after 16 days and died in 21 to 29 days, respectively. Four more received 0.25 cc of a 20 per cent suspension of spleen removed from a dog suffering from distemper (Lederle strain). Of these, three died in 17, 25 and 22 days, respectively, all having become very ill after 16 days. The fourth became ill also, but recovered.

Each of the four samples of unconcentrated serum (20 cc) was administered together with 10 mg of Lederle desiccated virus to

four ferrets. Two died after one and two days, probably owing to the large volume of foreign serum. They were replaced one week later. These animals died 34, 27, 24 and 30 days after the date of injection, having become ill after 30, 16, 13 and 19 days, respectively.

There were two ferrets which received Lederle desiccated virus together with 4 cc of concentrated serum (Little). These died after 35 and 42 days, thus living somewhat longer than the other animals which succumbed. Two more were injected with the same volume of the same serum along with 0.25 cc of the spleen emulsion. One remained well throughout; its mate became ill but recovered.

There were three additional animals which were injected one week after the large group, *i. e.*, at the same time as the two replacements. These received 4 cc of concentrated serum (Little) at that time, then two days later they were given 4 cc more and 10 mg of Lederle desiccated virus. These were kept for 14 days in a building about 100 feet away from the one in which all the others were housed, but were fed and handled by the same person who cared for the latter. They were then brought into the room used for the larger number of ferrets, some of which were ill at the time. Two of them became sick in eight days, and died six and nine days afterward; the remaining one sickened after eleven days and died four days later.

There are too few animals to warrant making explanations of the events. Yet one may perhaps be permitted to speculate. It will be observed that all the serum-treated ferrets (both unconcentrated—20 cc—and concentrated—4 cc and 8 cc) which were inoculated with the desiccated virus as well as the two controls which received this preparation, died. Of the four controls receiving the spleen emulsion, three died; and of the two which received the same emulsion and 4 cc of concentrated serum, one became ill and recovered, and its mate remained well throughout. Whether the virus preparation injected played any part in these results is therefore open to question.

We are inclined to think that the deaths among serum-treated animals were due to exposure to sick controls rather than to a failure of the serum to "hold" the virus. The deaths of the three ferrets which received 8 cc of concentrated serum as against the survival of two out of four which were given half as much serum are perhaps owing to a failure to supplement a small amount of artificial immunity (produced by injection of serum and virus) with exposure to subinfective doses resulting from contact with

the disease from its inception. Such exposure would tend to fortify the postulated artificially produced resistance, whereas sudden exposure to the ferret virus at the height of the disease proves fatal. There is also the possibility that a large amount of serum may be disadvantageous as far as the serum-virus injection method is concerned. Little has encountered such an experience in vaccinating dogs, so that it would appear as if calibration of serum against virus is necessary. A third possibility is that the three ferrets receiving 8 cc of concentrated serum might have died even if they had not been brought into sudden contact with sick animals, since, at the time they were moved, the incubation period, as found in the large majority of the ferrets, had not expired (measured by the number of days between injection and the appearance of visible illness).

Regarding the deaths of the ferrets which were injected with different unconcentrated sera, it will be noted that one animal remained well for 30 days and died four days later. This animal lived a somewhat shorter time than either of the two which were given 4 cc of concentrated serum (35 and 42 days) but somewhat longer than any of the three which received 8 cc of the same serum (28, 29 and 31 days). Its mates died in 27, 24 and 30 days. We should have preferred to have four ferrets injected with each of the unconcentrated sera, but facilities for handling the animals were limited.

REFERENCES

[1]Dunkin, G. W., and Laidlaw, P. P.: Studies on dog distemper. I. Dog distemper in the ferret. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 201.
[2]Little, G. W., and Lentz, W. J.: Production of immunity to canine distemper serum concentrate-living virus, simultaneous injections. Preliminary report. Jour. A. V. M. A., lxxxiii (1933), n. s. 36 (3), pp. 405-409.

REPORT ON SERUM CONCENTRATE (LITTLE)*

By M. L. MORRIS, *Stelton, N. J.*

The control of canine distemper has always seemed to me to constitute the major problem in the safe hospitalization of dogs. In view of this opinion, I have directed my efforts to ascertain the most feasible methods for its prevention, control, diagnosis and treatment.

The serum concentrate, as described by Little and Lentz,[1] prompted me to give this method careful consideration prior to its adoption as a clinical procedure in my hospital.

*Presented at the first semi-annual meeting of the American Animal Hospital Association, New York, N. Y., August 17, 1934.

The data I presented at the American Animal Hospital Association meeting in Cincinnati, Ohio, constituted my preliminary observations.[2] This information was voluntarily obtained for my own particular use. After giving this matter considerable laboratory and clinical study, it was apparent that it possessed merit. A knowledge of the difficulties encountered by veterinarians in combating this infection prompted me to present my findings to the profession. This again was done voluntarily.

The introduction of homologous serum was a real step forward in the handling of canine distemper. Its improvement by Dr. Little is another marked advance. This product now has been carefully tested by several laboratories and a number of well-qualified small-animal practitioners. Reports would indicate that it is meeting with favor. Small doses, no shock, concentration, and simplified prophylaxis are some of its commendable features. As a curative, large doses in the early virus stage give results. Once the secondary infections are established, its efficacy is diminished. I feel that we can adopt this serum for use in our hospitals and practices and obtain satisfactory results with it for the purpose for which it is designed, namely, to control the filtrable virus of Carré. It is not a sure cure for all cases of canine distemper.

Dogs immune to the filtrable virus are still susceptible to the secondary bacterial infections and under conditions of lowered resistance may develop septic infections. It is still essential to differentiate the symptomatology of secondary invaders from the primary virus disease. We, as veterinarians, must still apply scientific knowledge to the handling of all phases of distemper if we are to be successful. A product such as serum concentrate will not do the job alone. Our knowledge of this product is not sufficiently complete to justify its unqualified endorsement or acceptance. Further evidence and field trials are necessary to place it on a sound scientific basis.

REFERENCES

[1]Little, G. W., and Lentz, W. J.: Production of immunity to canine distemper by serum concentrate-living virus, simultaneous injections. Jour. A. V. M. A., lxxxiii (1933), n. s. 37 (3), pp. 405-409.

[2]Morris, M. L.: Clinical and laboratory studies of the simultaneous use of serum concentrate (Little) and living virus for immunizing dogs against distemper. No. Amer. Vet., xv (1934), 8, pp. 32-37.

Waiting Room for Dogs

A special waiting room for dogs has been provided without charge by the Chicago Zoölogical Gardens, at Brookfield, Ill., since visitors are not permitted to bring dogs inside the inclosure.

Dog World.

AVIAN TUBERCULOSIS OF SHEEP*

By G. S. HARSHFIELD *and* LEE M. RODERICK
North Dakota Agricultural Experiment Station
Fargo, N. Dak.

It is a well-recognized fact that the three types of the tubercle bacillus are invasive when infection occurs in certain heterologous hosts. Ordinarily, the character and extent of the lesions which develop under those conditions, however, are benign and non-progressive. This is the rule with the avian infection, which is so common in hogs; but sometimes that type produces lesions of disease in an individual of a heterologous host species, which compare in extent with those which develop in the homologous host. The studies included in this paper deal with the identification of the avian type of infection in a number of cases of tuberculosis in sheep and transmission trials on sheep with material of avian origin.

Tuberculosis of sheep is exceedingly rare, so that a case is primarily a pathological curiosity, for it is not an economic problem. The significance of tuberculosis in sheep from the standpoint of the sanitarian, therefore, is negligible. The rarity of the disease in the United States is emphasized by the statistics of the Bureau of Animal Industry for the six-year period ending July 1, 1933, as shown in table I.

TABLE I—*Tuberculosis in sheep slaughtered under federal inspection.*

FISCAL YEAR	SHEEP AND LAMBS SLAUGHTERED	RETAINED FOR TUBERCULOSIS	CONDEMNED FOR TUBERCULOSIS
1928	12,983,841	1	1
1929	13,768,865	1	1
1930	15,306,899	5	5
1931	17,300,432	3	3
1932	18,659,941	5	4
1933	17,283,875	2	2

Hutyra and Marek[1] report that the condemnation of sheep for tuberculosis in the German abattoirs varied from 0.14 to 0.22 per cent for the period from 1904 to 1918.

The literature on tuberculosis contains but few references to the disease in sheep, in which the type of the infection was determined. M'Fadyean[2] examined two cases which involved the

*Published with the approval of the Director of the Experiment Station as Paper No. 8 of the Journal Series of the North Dakota Agricultural College. Received for publication, March 5, 1934.

mammalian type. Two cases reported by Jowett[3] were of bovine origin, as was the one case typed by Van Es and Martin.[4] Griffith[5] studied two cases of generalized tuberculosis in sheep, both of which were of the avian type.

Five cases of avian tuberculosis in sheep have now been examined and typed at this laboratory. The first case, which was received April 1, 1927, was reported by Schalk.[6] The second case mentioned by Schalk apparently was tuberculous but it did not prove infectious for animals. Four additional cases are reported in this paper. This study is a report on the avian tuberculosis project which was started in 1922.

The material was obtained from abattoirs under federal inspection, with the coöperation of Drs. John R. Mohler, H. W. Schoening, J. H. Kitzhofer, J. A. Thompson, H. D. Osborne and L. M. Henry, of the Bureau of Animal Industry. One specimen (L. N. 28123) came from Kansas City, Mo., while the remainder were obtained from the plant at West Fargo, N. Dak. An emulsion of the lesions in salt solution was prepared by grinding the material with sand in a mortar and filtering through cotton. The suspected material was injected intravenously into chickens and intraperitoneally into cavias and rabbits. Young pullets were used, except for L. N. 28123, when it was necessary to use yearling birds. All of the birds had given at least two negative tuberculin tests prior to injection. We had no success in producing infection in rabbits with material from the sheep, but cultures 25141 and 26962 were quite pathogenic. The animals were allowed to live for three months, unless they died in the meantime except in one special case.

L. N. 25141.

The viscera of a three-year-old ewe, which was in good flesh, were received August 27, 1931.

Lesions: Extensive tuberculous lesions were found in the liver, lungs, spleen, mesenteric and body lymph-glands and on the pleura and peritoneum. The liver was enlarged with tumor-like masses on the surface. It was pale in color, firm in consistency and cut with difficulty because of a marked increase of connective tissue. Innumerable subpleural tubercles up to 8 mm in diameter were present in the lungs. The cut surface revealed an extensive, generalized, miliary tuberculosis. Many of the lymph-nodes were enormous in size, with caseation and calcification.

Microscopic examination of the lung revealed an acute miliary tuberculosis with acid-fast bacilli present. The tubercles were small, uniformly distributed, with little caseation or calcification,

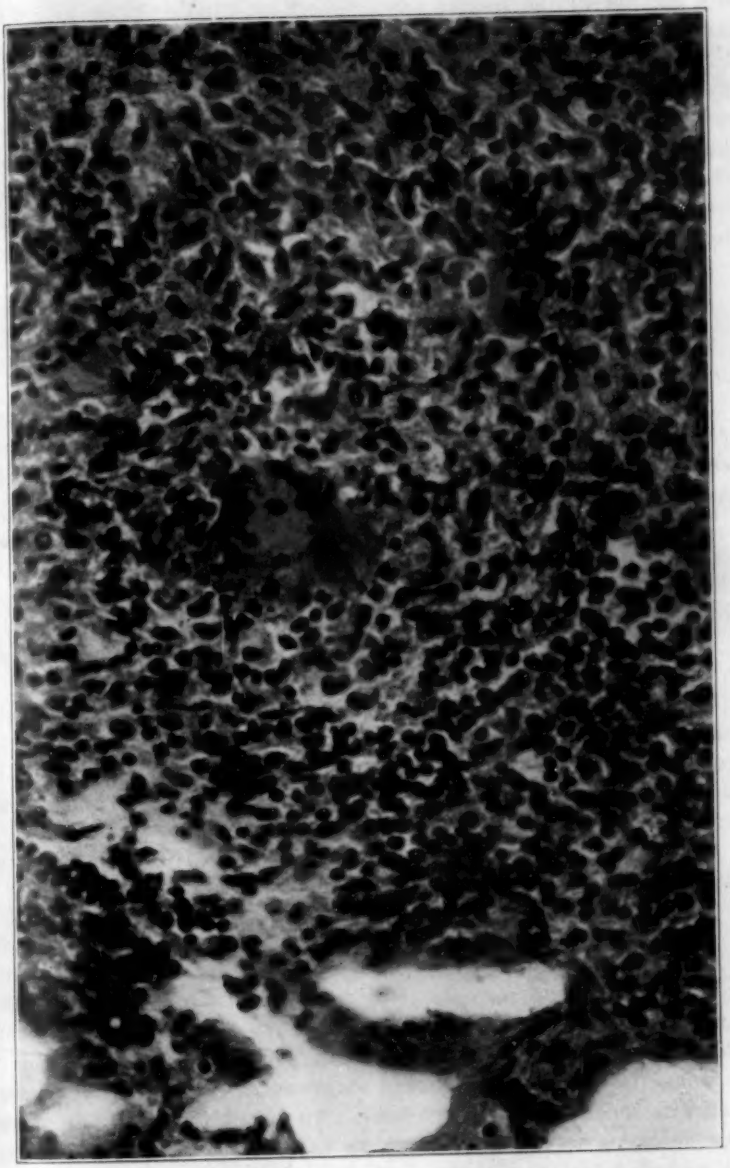

FIG. 1. Portion of a miliary tubercle of the lung of sheep 25141.

while giant-cells were numerous. We counted about 50 tubercles in a section 7 x 20 mm in size. There was little organization or exudation in the lung, while the process was older in the liver.

Inoculation results: Two young chickens and two cavias were inoculated with the material. One chicken died in 39 days with generalized miliary tuberculosis. The other showed extensive tuberculosis when killed. One cavia died shortly after injection, while the other was negative when killed. Material from these chickens was inoculated into another series and used in transmission trial I.

Infection type: A culture of the avian type was isolated.

L. N. 26962.

The viscera of a mature ewe, in good flesh, slaughtered September 22, 1932, were secured some days later.

Lesions: Varying degrees of caseation and calcification were present in the bronchial, mediastinal, portal, cervical, mesenteric, renal, hepatic, crural, popliteal, prescapular and pharyngeal lymph-nodes.

The lungs showed numerous yellowish tubercles beneath the pleura, uniform in distribution and up to about 0.3 cm in diameter. The bronchial and mediastinal glands were much enlarged, fibrotic, with much caseation and calcification.

The tubercles in the liver were fewer in number but similar in size to those in the lungs.

Inoculation results: Six chickens, two cavias and two rabbits were inoculated with material from the sheep. Four of the chickens developed miliary tuberculosis, while one died prematurely and the other revealed no gross lesions. The rabbits and cavias remained negative.

Infection type: A culture of the avian type was isolated from two of the birds on Petroff's medium.

L. N. 28123.

Portions of lung, spleen and lymph-gland of a sheep received July 27, 1933. Lesions resembling tuberculosis were present in the lungs and adjacent lymph-nodes, spleen, peritoneum and liver. Calcified lesions about 2 mm in diameter were found in the lung.

Inoculation results: A filtered emulsion of portions of the specimen was inoculated into two cavias, two rabbits and six chickens. The chickens were killed after an interval of three months and all revealed lesions of tuberculosis. The tubercles in the liver and spleen were few in number but uniform in size. The rabbits and guinea pigs were normal.

FIG. 2. Lungs of sheep 26962.

Infection type: Cultures isolated from the birds on Petroff's medium, were typical of the avian type.

L. N. 28341.

The viscera of an aged sheep slaughtered on October 4, 1933.

Lesions: The spleen was enormously enlarged, measuring 16 x 13 x 5 cm and weighing 610 gm. It was covered with fibrous adhesions, while the cut surface showed little splenic tissue, for it was a fibrous, calcified and caseated mass which was cut with difficulty.

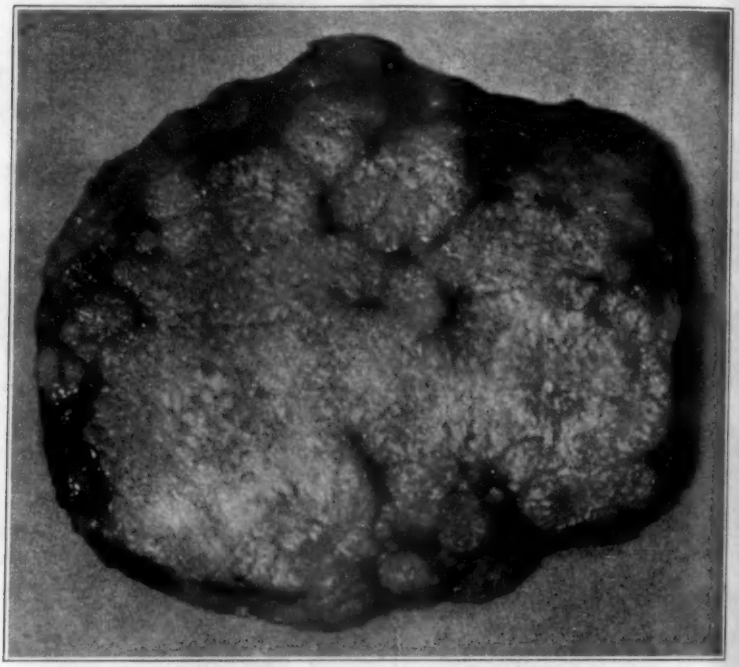

FIG. 3. Tuberculous spleen of sheep 28341.

The cut surface of the liver showed many tubercles from small white miliary foci to large fibrous, caseocalcareous masses, several centimeters in diameter. There were many tuberculous masses a centimeter or more in diameter under the diaphragm. Many glistening white tubercles were seen under the pleura and were evenly distributed through the deeper portions of the lungs. There was little caseation. The posterior mediastinal lymph-gland was a large fibrous and calcified mass measuring 2.5 x 13 cm.

Inoculation results: Four chickens and three cavias were inoculated with material. One chicken died with miliary tuberculosis of the liver and spleen after 30 days. The three other birds were killed after an interval of 43 days and all showed numerous tuberculous nodules in the liver and spleen. Two of the cavias were negative when killed, while acid-fast bacilli were present in the localized abscess at the point of inoculation in the third.

Infection type: A culture conforming to the avian type was isolated from the chickens.

Three interesting features of these four cases of bovine tuberculosis are worthy of note: (1) All were of the avian type of infection. (2) All were generalized. (3) All showed an extreme degree of calcification. There is little analogy in the behavior of the avian infection in sheep and hogs, two of its heterologous hosts. Infection readily occurs in the hog but it ordinarily is localized in character. This infection is difficult to produce in sheep and probably is regressive in character except in these rare individuals. We have seldom seen more extensive tuberculosis in any other animal than these sheep, nor have we seen such extensive calcification, for some of the lymph-glands could hardly be cut with the knife.

Transmission Trials

It was impossible to trace these cases to the possible contact with tuberculous fowls although avian tuberculosis is common in this area. Four transmission experiments were started, therefore, in order to determine if sheep could be infected with any degree of regularity. Griffith[5] was able to produce generalized tuberculosis in two lambs and two goats by subcutaneous inoculation of an avian strain isolated from one of his cases. In another attempt[8] he produced a chronic infection in the udder of a goat, by subcutaneous inoculation of an avian culture, which persisted for nearly five years.

All the lambs used in our transmission trials were challenged with both avian and bovine tuberculins before starting them on experiment and were subjected to both tests at monthly intervals until slaughtered. Intradermal tuberculins prepared in our laboratory were used for the tests. Injections were made into the skin of the caudal folds and the readings of the test made at the 72nd hour. At the time of slaughter, lymph-glands of the head, thoracic and abdominal cavities were collected from each sheep, ground in sterile mortars and an emulsion made with physiological saline solution. After filtration through cotton, the filtrate was injected into tuberculosis-free chickens to determine

the presence of viable tubercle bacilli. Chickens were injected regardless of whether or not gross lesions were found in the sheep at autopsy.

Experiment 1: Three spring lambs (90, 91 and 92) were injected intravenously with a filtered normal saline emulsion of the tuberculous liver and spleen of a chicken infected with sheep strain 25141, Nov. 16, 1931. The tuberculin reactions of this group are set forth in table II.

TABLE II—*Tuberculin reactions of three lambs intravenously infected.*

MONTH	LAMB 90		LAMB 91		LAMB 92	
	AVIAN	BOVINE	AVIAN	BOVINE	AVIAN	BOVINE
1931						
December	−	−	−	−	+	−
1932						
January	++	−	++	−	+++	−
February	++	−	+	−	+++	−
March	+++	−	+++	−	+++	−
April	+++	−	+	−	+++	−
May	+++	−	±	−	+++	−
June	+++	−	+		Slaughtered 5–18–32	
July	Slaughtered 6–6–32		±	−		
August			−	−		
September			±	−		
October			−	−		
November			−	−		
December			±	−		
1933						
January			−	−		
February			++	−		
			Slaughtered 3–27–33			
Infection	Positive		Negative		Slight	

Key: − = negative. ± = trace. + = slight. ++, +++ = well marked.

All three lambs gave well-marked reactions to avian tuberculin the second month following injection. In lambs 90 and 92 the reaction was strong on succeeding monthly tests. After the fourth month, lamb 91 began to show less sensitization and gave but one good reaction in later tests. No sensitization to bovine tuberculin was obtained on any test.

Lamb 92 was slaughtered, May 18, 1932, but no well-defined gross lesions of tuberculosis were found. Two of the chickens which were injected with the lymph-gland material died early and

were negative, but a slight infection was found in two killed out at three months. The lymph-glands evidently contained viable organisms in this case although no visible lesions were found.

Lamb 90 was slaughtered, June 6, 1932, and this was a case of generalized miliary tuberculosis. The lungs, liver, spleen, kidneys and many regional lymph-nodes were tuberculous. The mediastinal, mesenteric and renal lymph-glands showed distinct caseation and calcification. Four chickens injected with material from this sheep all developed tuberculosis.

Lamb 91 was killed, March 27, 1933, sixteen months after injection. Caseocalcareous lesions were seen in the bronchial, mediastinal, mesenteric and hepatic lymph-nodes. Four chickens were injected with this lymph-gland material but none developed the disease. The infection probably had been eliminated.

Experiment 2: Three spring lambs (93, 94 and 95) received ten feeds of ground livers and spleens of tuberculous chickens in the three-month period from November 16, 1931, to February

TABLE III—*Tuberculin reactions of sheep which received feeds of tuberculous chicken livers and spleens.*

MONTH	SHEEP 93		SHEEP 94		SHEEP 95	
	AVIAN	BOVINE	AVIAN	BOVINE	AVIAN	BOVINE
1931						
December	−	−	−	−	−	−
1932						
January	++++	−	++++	−	+++	−
February	+++	−	+++	−	+++	−
March	++	−	+++	−	++	−
April	+++	−	+	−	+++	−
May	+++	−	+	−	++	−
June	Slaughtered 5–27–32		+	−	+	−
July			±	−	−	−
August			+	−	+	−
September			−	−	−	−
October			±	−	−	−
November			−	−	−	−
December			±	−	−	−
1933						
January			+	−	+	−
February			±	−	+	−
March			±	−	±	−
			Slaughtered 3–27–33		Slaughtered 3–27–33	
Infection	Positive		Slight		Negative	

Key: − = negative. ± = trace. + = slight. ++, +++, ++++ = well marked.

19, 1932. The livers and spleens were ground and made into an emulsion with salt solution and mixed with the grain feed. The tuberculin reactions of this group are set forth in table III.

All three lambs showed well-marked avian reactions, beginning on the second month after starting the feeds. Lamb 93 was slaughtered while still showing a strong sensitization. In lambs 94 and 95, the degree of reaction became less within a few months and never gave more than a slight reaction afterwards. No reactions were obtained with bovine tuberculin.

Lamb 93 was slaughtered, May 27, 1932. Extensive caseocalcareous lesions of tuberculosis were found in the mesenteric lymph-glands. Practically every node in the chain was involved. No lesions were found involving the remainder of the viscera. Chickens injected with lymph-gland material from this case developed the disease in the liver and spleen. Isolation of the organism was made from the mesenteric lesions on Petroff's egg medium.

Lamb 94 was slaughtered, March 27, 1933, and slight caseocalcareous lesions were found in a mesenteric lymph-node. No other lesions were found. Two chickens injected with glandular material developed slight tuberculosis in liver and spleen; a third was negative.

Lamb 95 was slaughtered, Mar. 27, 1933. No gross lesions were found and the chickens injected with glandular material were negative.

Experiment 3: Six spring lambs (83, 84, 85, 86, 87 and 89) were placed in the cage barnyard, described by Schalk,[7] with a flock of tuberculous chickens on Oct. 20, 1931. Two additional lambs (56 and 57) were added on Feb. 2, 1932. During the winter months, these sheep were stabled with the continuous exposure maintained by the addition of the droppings from the tuberculous fowls to their feed. The avian tuberculin reactions of this group are set forth in table IV. Tests with bovine tuberculin were also run in conjunction with the avian test but they were uniformly negative.

All of the lambs in this group with the exception of lamb 89 gave a positive reaction to avian tuberculin within a few months after the beginning of the exposure, but the sensitization was of short duration. Lamb 87 gave a strong reaction on three successive tests and was the only animal to show more than one good reaction.

Sheep 57 died on October 7, 1932, of a ruptured bladder from urinary calculi. A few small caseocalcareous lesions were found

TABLE IV—*Avian tuberculin reactions of the group of lambs exposed to a flock of tuberculous chickens in experiment 3.*

MONTH	LAMB							
	83	84	85	86	87	89	56	57
1931								
November	−	−	−	−	−	−		
December	−	−	−	−	−	−		
1932								
January	−	−	−	−	−	−		
February	−	−	++	++	+++	−		
March	+++	++	±	−	+++	−	−	−
April	−	+	−	−	+++	−	+	+++
May	−	−	−	−	+	−	+++	+
June	−	−	−	−	−	−	+	+
July	−	−	+	−	−	−	−	−
August	−	−	±	−	−	−	−	−
September	−	−	−	−	−	−	+	−
October	−	−	−	−	−	−	−	−*
November	−†	−†	±†	−†	−†	−†	−†	
Infection	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.

Key: − = negative. ± = trace. + = slight. ++, +++ = well marked.
*Lamb 57 died, October 7, 1932.
†Slaughtered, November 2, 1932.

in the mesenteric lymph-glands but the chickens injected with the material remained negative for tuberculosis.

The remaining animals of this group were slaughtered, November 2, 1932, slightly over a year from the beginning of the exposure. Gross lesions were absent except in lambs 83 and 84. A small focus was found in the mesenteric lymph-gland of one and in the mediastinal of the other.

Chickens were injected with lymph-gland material from each of the lambs in this group but an outbreak of infectious laryngotracheitis killed a large number of the birds before an infection could have been established. No tuberculosis was found in the birds which survived the outbreak.

Experiment 4: A group of five spring lambs (108, 109, 113, 114 and 120) were exposed to a flock of tuberculous chickens, starting November 7, 1932. Two additional lambs (127 and 128) were placed with this group on January 16, 1933. The exposure was established in the same manner as in experiment 3. The results of the avian tuberculin tests on this group are given in table V. The bovine tuberculin tests were consistently negative.

This group of lambs showed a greater degree of sensitization to avian tuberculin than those in experiment 3, but in each instance was lost or became a mere trace in later tests. They were

TABLE V—*Avian tuberculin reactions of the group of lambs exposed to a flock of tuberculous chickens in experiment 4.*

MONTH	LAMB						
	108	109	113	114	120	127	128
1932							
December..	−	−	−	−	−		
1933							
January....	−	+++	−	±	−		
February...	−	+++	+++	+++	−	−	−
March.....	++	+	+++	++	+++	−	−
April.....	−	−	+++	−	+++	++	+++
May.....	+	−	+	−	+	++	+++
June.....	−	−	++	−	−	++	+++
July.....	−	−	−	−	+	−	+++
August....	+	−	+	−	+	+	+++
September..	±	±	±	−	±	−	+
October....	−	+	−	−	−	±	+
Infection*..	Neg.	Neg.	Slight	Neg.	Neg.	Slight	Neg.

Key: − = negative. ± = trace. + = slight. ++, +++ = well marked.
*All lambs in this experiment were slaughtered, October 27, 1933.

slaughtered, October 27, 1933, almost a year after beginning exposure.

No gross lesions suspicious of tuberculosis were found in this group except in lamb 127. Small caseocalcareous lesions were seen in the mesenteric lymph-glands. Chickens were injected intravenously with a filtered saline suspension of lymph-gland material from each lamb and a slight tuberculous infection was produced in the birds receiving material from lambs 113 and 127. The others remained negative.

A total of 18 sheep were exposed to avian tuberculosis by the ingestion route and three by intravenous injection. All of the sheep in these transmission trials were no doubt given a greater exposure than farm flocks would receive, yet only six of the animals carried the infection at the time of slaughter, as evidenced by the results of chicken inoculation trials with lymph-gland material. Four of the positive cases had received a massive dose of infective material, two by the intravenous route and two by numerous feeds of infected chicken livers. In addition, three of these animals were slaughtered before the sensitization to avian tuberculin began to fade. It is quite possible that we would have had more positive cases had they all been slaughtered while strongly sensitized, for the tuberculin reactions would indicate that the sheep which were kept for the longer period were capable of eliminating the infection responsible for the sensitization to tuberculin. Four lambs (91, 83, 84, 57) contained what

appeared to be gross lesions of tuberculosis on postmortem examination; yet no infection was produced in chickens injected with the material. This again may be evidence that the sheep has a remarkable ability to overcome a tuberculous infection.

One generalized case of tuberculosis was produced in sheep 90 which had received an intravenous injection of a filtered emulsion of a tuberculous chicken liver. This strain was from one of the original sheep.

Some of the recent work on the dissociation of the tubercle bacillus by Sabin *et al*[9] and Seibert *et al*[10] suggested at least that these rare cases of tuberculosis in sheep might have been due to mutations or variants of unusual virulence. We have been unable to confirm this assumption. The infected chickens have regularly shown positive tuberculin reactions which might have been absent if infection had occurred with the more virulent smooth variants. The examination of sections of the livers and spleens of chickens, six weeks after inoculation, with the cultures of the ovine strains as well as one set of chickens inoculated with the original material, revealed many bacilli and yet caseation of the tubercles was quite evident. The unusual character of the lesions in these sheep is a paradox in pathology but seems to represent the reaction of a heterologous host to an infection which is not invasive enough to produce gross disease except in rare individuals or with overwhelming amounts of the infection.

Summary

Four cases of avian tuberculosis in sheep, which developed under field conditions, are reported. All were generalized cases with extensive dissemination of the disease and marked calcification of the lesions.

Three test sheep, injected intravenously with one of the strains, resulted in two positive cases, one of which was generalized. Two positive cases were produced by the feeding of tuberculous chicken livers. The disease was localized in the mesenteric glands in both. Fifteen sheep were exposed to a tuberculous chicken flock for approximately one year and this exposure resulted in infecting two of that number. Sensitization to avian tuberculin was produced in 20 of the 21 experiment sheep, although the period of sensitization was short. No reactions were obtained to mammalian tuberculin. Those transitory infections were, therefore, no doubt regressive in most cases.

Report of Two Additional Cases

Since the preceding material was prepared for publication, an opportunity was given us, through the courtesy of Drs. H. W.

Schoening and L. Enos Day, of typing two additional cases of suspected tuberculosis in sheep.

L. N. 28992.

Portions of the lungs, liver and a lymph-gland of a sheep were received April 16, 1934. The sheep was slaughtered in New York, N. Y. Small foci of pinhead size were distinguishable in the lymph-gland and a small caseous tubercle was present in the lung specimen.

Inoculation results: A filtered emulsion of portions of the specimens was injected into four chickens and three cavias. One chicken died 52 days after injection with miliary tuberculosis of the liver and spleen and another was killed after 78 days which showed similar lesions. The remaining fowls were also positive for tuberculosis when killed after three months. The tubercles were not numerous but were of uniform size in the liver and spleen. Smears of the lesions contained large numbers of acid-fast bacilli. The guinea pigs remained normal.

Infection type: Avian.

L. N. 29038.

Specimens of lung, liver and adjacent lymph-glands of a sheep slaughtered at Wichita, Kan., were received on April 27, 1934. The liver and lung tissues were seeded with small tubercles, ranging up to 0.5 cm in diameter. Calcification was not noticeable except in the adjacent lymph-glands.

Inoculation results: Four chickens and three cavias were injected with a filtered emulsion of the tissue specimens. Two of the chickens killed after 67 days were positive for tuberculosis, as were the remaining two killed 82 days after injection. The livers and spleens of all the birds were seeded with large numbers of tubercles of uniform size. Acid-fast bacilli were numerous. None of the guinea pigs developed tuberculosis.

Infection type: Avian.

This makes a total of seven positive avian type cases of ovine tuberculosis typed at this laboratory, six of which are included in this report.

REFERENCES

[1]Hutyra, F., and Marek, J.: Spezielle Pathologie u. Therapie der Haustiere. (6. Aufl., Bd. 1, 1922), p. 580.
[2]M'Fadyean, J.: Cited by Griffith, A. S.: Jour. Comp. Path. & Therap., xxxviii (1925), p. 157.
[3]Jowett, W.: Jour. Comp. Path. & Therap., xli (1928), p. 255.
[4]Van Es, L., and Martin, H. M.: Neb. Agr. Exp. Sta. Res. Bul. 49 (1930).
[5]Griffith, A. S.: Jour. Comp. Path. & Therap., xxxviii (1925), p. 157.
[6]Schalk, A. F.: Jour. A. V. M. A., Lxxiv (1929), n. s. 27 (3), pp. 301-311.
[7]Schalk, A. F.: Jour. A. V. M. A., Lxxii (1928), n. s. 25 (6), pp. 852-863.
[8]Griffith, A. S.: Jour. Comp. Path. & Therap., xliv (1931), p. 144.
[9]Sabin, F. R., Miller, F. R., Doan, C. A., and Wiseman, B. K.: Jour. Exp. Med., liii (1931), p. 51.
[10]Seibert, F. B., Long, E. R., and Morley, N.: Jour. Inf. Dis., lii (1933), p. 175.

OBSERVATIONS ON THE SHEEP TAPEWORM, MONIEZIA EXPANSA, IN CALIFORNIA*

By STANLEY B. FREEBORN *and* LESTER J. BERRY
University of California, Davis, Calif.

The practical sheepmen of California during the past few years have become alternately alarmed and reassured concerning the dangers from liver flukes and stomach worms (*Ostertagia*) as these parasites became first important and eventually negligible through the application of simple control methods. The reaction to these parasites in a majority of the sheep-raising areas of California is one of tolerance. Some growers realizing that methods are at hand for eliminating their losses prefer to postpone control and take their losses while others exercise careful control and reap the benefits derived from parasite-free sheep.

Recently, however, many inquiries have been made and considerable concern has been felt in connection with the common sheep tapeworm, *Moniezia expansa* Rud. Infestation with this parasite is almost universal in California sheep but the individual infestation is of low grade, three or four worms constituting the average infestation.

To determine the effect, if any, of this parasite on growing lambs, a group of 24 purebred Shropshire lambs, three months old, were segregated, by means of fecal examinations, into four groups. One group was made up of positive controls, another the negative controls, and the two other groups were positive for tapeworms but treated at the beginning of the trials with Cunic mixture as described later to remove these parasites. Naturally some of the negative controls became positive during the course of the trials and at the end of 77 days of trial two of the positive controls had become negative naturally. In one type of treatment all the positive lambs were rendered negative and remained so to the end of the trial and in the other type only about half were rendered negative by treatment. After shifting these aberrant cases to their proper classification, we found that the positive cases which remained positive for the 77 days of the trials averaged to make a weekly gain of 2.82 pounds, as contrasted with 2.84 pounds for the negative cases — a negligible difference.

Inasmuch as the market value of lambs within a given weight class is largely a matter of condition, we obtained the services of

*Received for publication, March 12, 1934.

a particularly competent judge to grade the lambs at the expiration of the trial into the five accepted market classes: prime, choice, good, medium and common. By giving a numerical value of 5 to the prime lambs, and decreasing the value by one for each succeedingly less valuable class to a value of one for the least desirable, we arrived at the following results in which a perfect score would be 5:

Positive controls	2.16
Those made negative by treatment	2.66
Negative controls	4.33

Naturally this evaluation was based on the judgment and experience of the judge, but granting that it was correct, it means not only that the lambs that had not been subjected to tapeworms were more pleasing in appearance and condition but that they were more valuable to the buyer. In other words, the lambs that were continually free of tapeworms had made better use of their food to form "finished" lambs while those harboring the parasites, although making the same weight, were "coarse" lambs less economical for slaughter.

Attempts to Produce Infestation

Sinitsin[1] has proposed that the individual onchospheres under optimum conditions continue to develop outside the body and eventually produce two elongate arms which become coiled about each other, thus furnishing the motive power to "unwind" in the presence of a moist membrane and become attached or invade the tissue when the capsule is ruptured in the nasal passages, the mouth, or throat. Although we have observed thousands of these embryos under varying conditions of moisture and dryness, we have never observed this complicated arrangement proposed by this author. The lengthening of the arms and the other manifestations noted by Sinitsin have been observed.

To condense material from lengthy protocols, we will merely state that we used these incubated onchospheres in every fashion that we could conceive with muzzled, susceptible lambs for two years without convincing ourselves that we had produced in this fashion a single positive case. The onchospheres were incubated for periods of from a single day to over a year; they were kept moist and dry as well as alternating these conditions; they were placed in the mouth and blown into the nostrils but in only one case did we obtain any evidence of interest. In our first series of experiments in 1932, four crossbred lambs two months old and negative by fecal examination for tapeworms were confined in a

new box-stall with controls and fed approximately 500 onchospheres that had been incubated in a moist chamber for three weeks. They were fed cows' milk but were not muzzled. At autopsy, 44 days later, all the controls were negative and three of the treated animals, but in the lumen of the jejunum of the fourth lamb we found an interesting cysticercoid larva.

This larva (fig. 1) was shaped like a liver fluke but the leaf-like sac was swollen with a clear fluid to a thickness equal to half its width. The scolex end was roughly spherical and showed a wide transverse slit which opened and closed in salt solution, disclosing a rounded structure within which moved forward as the lips of the slit opened. No attempt was made to dissect out what was undoubtedly the scolex, as it was thought best to pre-

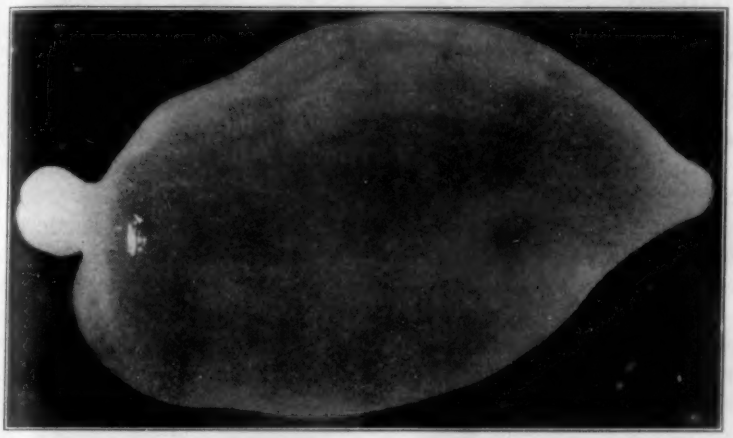

FIG. 1. Larva of *Moniezia expansa*.

serve the specimen entire and depend on clearing to differentiate the enclosed structure. The sac cleared very nicely but all attempts to clear the scolex portion were in vain and we have been able to discern only the rough outline of an enclosed scolex without any details. The attached sac was leaf-like, 15 mm long and 6 mm wide at the shoulders, tapering to a point behind and swollen to a thickness of 3 mm by a clear fluid. The walls were thin, membranous, and the entire surface marked with minute transverse striations. No hooklets were present in the sac.

In the absence of any anoplocephalid larvae for comparison, we are naturally loathe to suggest that this larva is that of *Moniezia expansa*, but by a process of elimination it seems at least worth mentioning as a possibility. Neither do we wish to

leave the impression that this larva resulted from the ingestion of the dose of onchospheres previously administered, as the elapsed time was too great, judging from field experience. The complete absence in the other treated animals would suggest that it was a chance infestation brought into the stall on bedding or the feet of attendants. However, if this larva is actually that of *Moniezia expansa*, it does verify the fact that the parasite is capable of utilizing the lamb's intestine for that stage of its development for which nearly all other tapeworms require a separate intermediary host. The general failure of direct feeding trials coupled with this observation suggests that possibly the missing factor is one of properly conditioning the onchospheres (by passage through the alimentary tract of an invertebrate?) before ingestion by the primary host.

IMMUNITY

Seddon[2] points out that lambs that have recovered from *Moniezia* infestations are immune to reinfestation. He points out[3] also that the duration of the parasitic infestation is about 65 to 70 days, 40 to 50 days of which are required to reach maturity and only 20 to 30 days of which are occupied in depositing eggs. From the data contained in his reports it is evident that he dealt with much heavier infestations than we have ever found in California sheep which may account for some of the discrepancies with our work. Our positive control group mentioned in the experiment to determine the effect on growth were already voiding eggs on April 5, when the experiment was started, and weekly fecal examinations showed some of these animals continuously positive for nine months. This author also points out that under Australian conditions no sheep over 18 months of age are ever found to be infested but assumes that this is not an age resistance but an immunity acquired by previous infestation. We are not able to point out a case of reinfestation following a naturally terminated infestation but three of our six lambs that were freed from *Moniezia* in April with a copper-nicotin drench and remained free from infestation throughout the following spring, summer and fall became reinfested, voiding ova for the first time in late December, nine months after treatment.

Under California conditions it is not at all unusual to find moderate infestations of *Moniezia* in bucks and ewes three and four years old, and on one occasion positive feces were obtained from a band of old ewes that had all lost their teeth, thus assuring that they were at least seven years old. Some immunity is developed, however, for during the summer and fall that our

treated animals remained free from infestation, undoubtedly protected by their immunity, other animals that had never been positive were continually becoming infested while grazing on the same pasturage.

Super-infestation is undoubtedly impossible even under our conditions of moderate parasitism, for in hundreds of autopsies the development of the tapeworms present appears to be equal. In addition, the normal disappearance of the tapeworms by death occurs in most cases almost simultaneously in a band that were lambs together and consequently subjected to infestation at the same time.

Our only suggestion is that the relatively light infestations of California sheep produce a degree of immunity capable of preventing super-infestation but of only a few months duration after the original infestation has been eliminated.

TREATMENT

The use of a combination of copper sulfate and nicotin sulfate for tapeworm treatment is evidently traceable to Dr. Cooper Curtice, of the U. S. Bureau of Animal Industry, and passed on to the writers by Dr. Maurice C. Hall, of the same bureau, by word of mouth. This combination, which Dr. Curtice called the "Cunic" mixture, consisted of water to which was added 1 per cent (weight) of copper sulfate and 1 per cent (volume) of nicotin sulfate (Black Leaf 40). The dosage for a full-grown ewe was four ounces graduated to two ounces for lambs three months old.

Although neither of these materials given alone is effective for tapeworms, the combination mentioned above is apparently a specific. Fecal examinations of over 200 individuals before and after treatment have failed to demonstrate a single case that did not show perfect control. Bliss[4] has shown that as a stomach poison for mosquito larvae a nicotin-copper combination is more toxic than nicotin alkaloid and inasmuch as the increased toxicity far outweighs the toxicity of copper, he assumes that a new compound is formed. This is borne out by the fact that a 1 per cent solution of Black Leaf 40 without the addition of copper sulfate is decidedly toxic in a four-ounce dose, whereas the addition of the copper renders it harmless even to weakened animals.

According to Aumeras and Tamiseier,[5] in a nicotin-copper complex only the nitrogen of the pyridine radical reacts with the copper ion giving a dissociation constant of 2.2×10^{-3}. As dissociation increases with dilution, it is hard to conceive of the Cunic mixture, already prepared in a weak dilution and further diluted with stomach contents, functioning as anything except the

separate copper and nicotin ions which we know to be ineffective against tapeworms. The first alternative would seem to be that possibly the copper combines with the impurities in Black Leaf 40 which we used as our source of nicotin sulfate. It is practically impossible to eliminate the nicotin from Black Leaf 40 without altering the impurities, so we attacked the problem backhandedly by treating a group of infested ewes with a drench made with 1 per cent copper sulfate and 0.4 per cent nicotin alkaloid, thus eliminating from the mixture the impurities of the Black Leaf 40. The results substantiated the above suggestion as there was apparently no effect on the tapeworms. In addition, the mixture, which contained no more nicotin alkaloid than the standard Cunic mixture made with Black Leaf 40, was decidedly toxic to the ewes.

The ease of administration of a dry powder in capsule form over a dilute liquid drench led us to the attempt to treat with a combination of Black Leaf 40 and copper sulfate to form a powder in dosages equivalent to the amount received in four ounces of the drench. This combination is a semi-liquid mixture, and inert fillers were used to dry the material after which it was administered in capsules. The results were disappointing, as only 50 per cent of the treated positive lambs were completely freed of their parasites, as contrasted with the perfect results of the drench treatment.

We next combined a 1-1 molar mixture dilution of nicotin as Black Leaf 40 with anhydrous copper sulfate. This solidifies as a green crystalline mixture almost immediately. This was powdered and given to infested ewes in doses of 1.8 grams of the mixture per animal. Each dose contained 1.28 grams of Black Leaf 40 and 0.554 grams of anhydrous copper sulfate. This treatment resulted in a complete removal of the tapeworms from the four ewes that received the preliminary treatment, but a band of infested lambs treated in the same way gave only a little better than 50 per cent efficiency as judged by the number of animals completely freed from tapeworms. Further trials with this type of treatment which are now in progress should yield results comparable with the drench treatment with the Cunic mixture.

REFERENCES

[1]Sinitsin, D. F.: A glimpse into the life history of the tapeworm of sheep, *Moniezia expansa*. Jour. Parasitol., xvii (1931), pp. 223-227.
[2]Seddon, H. R.: The development in sheep of immunity to *Moniezia expansa*. Ann. Trop. Med. & Parasitol., xxv (1931), pp. 431-435.
[3]Seddon, H. R.: On the life of *Moniezia expansa* within the sheep. Ann. Trop. Med. & Parasitol., xxv (1931), pp. 437-442.
[4]Bliss, A. H.: Toxicity studies of nicotine and nicotine-copper complex to mosquito larvae and pupae. Unpub. thesis, Univ. Calif. (1933).
[5]Aumeras, and Tamiseier,: On the constitution and stability of two copper-nitrogen complexes. Chem. Res., cxcii (1931), pp. 1156-1158.

THE TOXICITY OF CERTAIN SPECIES OF CROTALARIA SEED FOR THE CHICKEN, QUAIL, TURKEY AND DOVE*

By E. F. THOMAS,† *Gainesville, Fla.*
Florida Agricultural Experiment Station

During December, 1931, a poultryman in the vicinity of Gainesville, Fla., lost 34 hens from an acute condition; 40 to 50 hens in the flock showed symptoms of illness. The litter used in the house in which this situation occurred consisted of hulls of *Crotalaria spectabilis* Roth, which contained numerous mature seeds. The litter had been put in the house a few days previously. *C. spectabilis* seeds were found in the crop and gizzard of dead birds and the postmortem lesions were similar in all cases. These seeds were suspected of being the cause of the losses.

Plant introduction files of the Florida Agricultural Experiment Station indicate that *C. spectabilis* was first received at the Experiment Station from the United States Department of Agriculture in March, 1921, as *Crotalaria sericea* under foreign seed and plant introduction No. 51839.[1] Since that time, this species of Crotalaria has been grown in most parts of the State and many of the other southern states where it is used as a soil-building legume.

CROTALARIA SPECTABILIS ROTH‡

Plant herbaceous, never woody, to 2 meters tall, erect, pubescent, green; branches erect, not crowded, coarse, terete but ridged below each leaf, finely pubescent; stipules lanceolate to triangular, persistent, green, pubescent on lower surface, to 2 cm long; leaves very numerous, simple, obovate, apiculate, slightly undulate, glabrous and dark green above, paler and finely pubescent beneath, 12 to 20 cm long, 5 to 9 cm wide; racemes terminal or from the upper leaf axils, simple, to 50 cm long, 20 to 40 flowered; bracts 8 to 15 mm long, green, broadly triangular; pedicels slender, 10 to 18 mm long; calyx campanulate 10 to 14 mm long, lobes longer than the tube, triangular, acute; corolla conspicuous, 20 to 24 mm long; standard 2 cm long, 2.5 cm broad, yellow, standard and wings striate with light purple lines; pod 4 to 5 cm long, 10 to 18 mm wide, inflated, glabrous, green, becoming almost black, beak declined, 4 mm long; seed numerous

*Received for publication, March 12, 1934.
†Resigned, July 1, 1933.
‡Description of plant prepared by Erdman West, Mycologist, Florida Agricultural Experiment Station.

on slender stalks, smooth but creased on each side, mottled becoming almost black, 5 mm long.

Flowers—June to October.

Formerly called *C. sericea* Retz.

Widely distributed in Florida.

Several species of Crotalaria have been reported as poisonous. Stalker[2] found *C. sagittalis* L. to be poisonous for horses and cows. Theiler[3] found the fresh green plant of *C. burkeana* Benth. would produce laminitis in cattle. The dry crotalaria plant did not produce laminitis. Theiler found *C. dura* Wood and Evans to cause sickness and death in equines, the lesions produced were pulmonary emphysema, pneumonia and parenchymatous hepatitis and cirrhosis. Watt and Breyer-Brandwijk[4] refer to a reported fatal poisoning in horses by *C. globifera* E. Mey. White[5] refers to work by J. C. Lewis where goats were killed by artificially feeding 2 to 3 ounces of fresh *C. striata* D. C. leaves. Lewis was of the opinion that it was an endothelial poison. A feeding trial using one sheep at the Gwebi Experiment Farm[6] with *C. juncea* L. seed produced weakness, a tucked-up condition of the abdomen, and intestinal catarrh that lasted until death, which occurred eleven days after feeding.

Since chickens and wild birds in Florida have access to large acreages of *C. spectabilis* and other species of Crotalaria, it was deemed advisable to conduct experiments to determine the toxicity of the various available species of Crotalaria for chickens and other birds.

Experimental Data

Chickens: Each of four birds, which were force-fed 20 *C. spectabilis* seeds, showed no symptoms of poisoning. Autopsy, 40 days after feeding, revealed no lesions.

No clinical symptoms of poisoning were noted in four birds which were force-fed 40 seeds. Upon postmortem examination 21 days later, two birds were apparently normal while the remaining two showed petechiae on the heart muscle and liver. Endocarditis also was present in one bird.

Each of a third group of two birds was force-fed 80 seeds. Death occurred 35 and 64 days, respectively, after feeding. One bird, force-fed 160 seeds, failed to show symptoms of poisoning, while a second bird fed a like amount died in four days. The latter showed lesions of acute poisoning while the former, destroyed 77 days after being fed, showed lesions of chronic poisoning.

Each of two birds, force-fed 320 seeds, died of acute poisoning, one on the third day after being fed and the other on the tenth day. One bird, force-fed 20 seeds daily, died in 16 days. A second bird, force-fed five seeds daily, died in 18 days. Two control birds remained normal.

Three birds on a regular ration of mash and scratch were given free access to *C. spectabilis* seed. These birds died showing typical symptoms and lesions of poisoning three days after being given access to the seed. Three birds were allowed access to *C. spectabilis* seed only. These birds died 9, 10 and 37 days after given access to the seed.

Another group of three birds was placed on a regular ration of mash with *C. spectabilis* seed in the litter. One bird died in four days; the remaining two birds were destroyed 83 days later and while no symptoms of poisoning were evident at this time, yet each showed lesions similar to those of birds of earlier experiments in which death was induced by feeding *C. spectabilis* seed.

A fourth group of nine birds received a regular ration of mash and scratch feed and were given access to a field containing ripe *C. spectabilis* seed. Two birds died showing typical symptoms and lesions in two and five days, respectively, after being exposed. Four of the remaining birds were destroyed 30 days after exposure and, while showing no symptoms of poisoning at this time, each showed lesions as found in chronic cases of poisoning. Three birds destroyed 90 days after exposure were similar to the four just described but the lesions were less intense.

Experiments were conducted on adult birds to determine if other species of Crotalaria were toxic when force-fed. *C. incana* L., *C. grantiana* Harvey, *C. striata* D. C., and *C. intermedia* Klotschy seed were force-fed in 5- to 10-gram doses to groups of two birds. These birds failed to show symptoms of poisoning and no lesions were found when these birds were destroyed for autopsy.

Quail: Experiments also were conducted to determine the effect of feeding definite numbers of *C. spectabilis* seed to 8-month-old quail.

Each of two birds force-fed 20 seeds did not show symptoms or postmortem lesions of poisoning. Two quail, force-fed 40 seeds, apparently became sick but did not show lesions considered to be typical of crotalaria poisoning. Two quail, force-fed 80 seeds, died in 24 hours and two quail, force-fed 160 seeds, died in 48 and 72 hours, respectively. These latter two groups of quail showed lesions of crotalaria poisoning.

Two quail were given access to *C. spectabilis* seed with their regular feed but did not become sick nor were lesions found upon autopsy. Six quail given access to a field of ripe *C. spectabilis* seed did not show any symptoms or lesions of poisoning.

Two quail, exposed to *C. spectabilis* seed in litter on the floor of their pens, failed to show symptoms of poisoning.

Turkeys: One turkey, force-fed 320 *C. spectabilis* seed, and one turkey, force-fed 1,000 such seed, the latter at the rate of 20 seeds

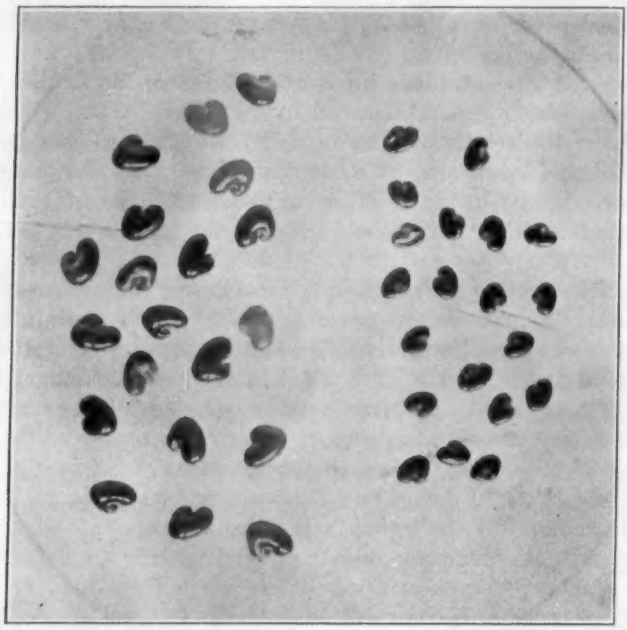

FIG. 1. Seeds of *Crotalaria spectabilis*. Left, swollen seeds recovered from crop and gizzard; right, normal seeds.

daily, failed to show symptoms of poisoning. When destroyed for autopsy, no lesions were found. Mr. T. W. Shuler, superintendent of the State Game Farm, Raiford, Florida, placed six turkeys from a wild strain in a field of *C. spectabilis* where ripe seed were in abundance. Ten days following the time they were placed in the field, an examination was made of the viscera from one turkey and no evidence of *C. spectabilis* poisoning was noticed; neither were there any seed found in the digestive tract. Mr. Shuler stated that the other birds were examined by himself when destroyed at a later date and they were all apparently normal and that no seed were found.

Doves: One dove, force-fed 80 seeds of *C. spectabilis*, and another dove, force-fed ten seeds daily for ten days, died of poisoning in 19 and ten days, respectively.

Discussion

There are two types of crotalaria seed poisoning, acute and chronic, depending on the amount of seed consumed and the resistance of the individual chicken. The acute type kills the birds in from one to ten days, but birds with the chronic type may linger for months. The symptoms in the acute type are: a full crop, congested comb, and sometimes a watery discharge from the mouth and nostrils. The symptoms of the chronic type may be the same as for the acute for the first few days. After this stage the comb loses the purple color and becomes scaly and the bird is unthrifty in appearance.

Upon postmortem examination, birds with the acute type will have crotalaria seed in the crop and gizzard, and these organs have the odor of crushed crotalaria leaves, the flesh is dark colored and small red hemorrhages occur in the fat around the heart and gizzard. Usually a jelly-like substance covers the liver and sometimes a fluid is found in the body cavity. Gas is sometimes noticed in the peritoneum where it attaches to the sternum.

With the chronic type the liver is about the only organ to show the effect of the poison. It is usually smaller and has rounded borders as though the sharp edges had been trimmed off. It is dull gray in color, and the capsule is thickened. It is often found to be adhered to adjacent organs.

During the time these experiments have been in progress, there have been several cases of poisoning by *C. spectabilis* seed reported by poultrymen and in a few cases the writer has autopsied some of the suspected birds and found typical lesions and *C. spectabilis* seed in the crop and gizzard. In all of these cases the birds secured the seed by feeding in fields of crotalaria. On one farm where *C. spectabilis* had been planted, it was decided to use the same area for chickens. The plants were destroyed by cutting the stalks. Chicks three to six weeks old were put on the area. A few of the chicks were found to be poisoned by the seed which were left in the soil four to six months after the stalks had been cut. These chicks showed typical lesions of crotalaria poisoning but somewhat more severe than in older birds. They showed enormous amounts of clear gelatinous exudate in the peritoneal and thoracic cavities, and the liver was atrophied and knotty in appearance, with numerous petechial hemorrhages over the liver surface.

Symptoms and postmortem lesions of *C. spectabilis* seed poisoning in quail differ somewhat from those found in chickens. Ruffled feathers, droopiness and sometimes diarrhea were about all the symptoms shown. Dark brown liver, which is sometimes streaked with pale areas, urates in kidneys and ureters, and crotalaria seed in crop and gizzard were the postmortem lesions found. The crop and gizzard have a distinct odor of crushed crotalaria leaves.

The chronic type of *C. spectabilis* seed poisoning was not observed in quail.

The postmortem lesions of *C. spectabilis* seed poisoning found in doves consisted of chronic pericarditis, enteritis, congested liver, and the contents of the cloaca greenish in color.

Conclusions

1. *Crotalaria spectabilis* Roth seed are toxic for chickens, quail and doves, when fed or eaten in considerable numbers.
2. Chickens will eat *C. spectabilis* seed under natural conditions, and sickness and death may be produced.
3. Quail did not eat *C. spectabilis* seed placed before them; neither did they eat them in the field.
4. Turkeys were not poisoned by as many as 1,000 *C. spectabilis* seed.
5. *C. striata* D. C., *C. grantiana* Harvey, *C. incana* L., and *C. intermedia* Klotschy seed were not toxic when force-fed in 5- and 10-gram doses to chickens and quail.

References

[1]Stokes, W. E.: Personal communication.
[2]Stalker, M.: Crotalism—A new disease among horses. Rpt. Iowa Agr. Coll. Dept. Botany (1884), pp. 114-115.
[3]Theiler, A.: Stiff-sickness or Stijfziekte in cattle. Agr. Jour. So. Africa, i (1911), pp. 10-21.
[4]Watt, J. M., and Breyer-Brandwijk, M. G.: The Medical and Poisonous Plants of Southern Africa. (E. and S. Livingstone, Edinburgh, 1932, pp. 72.)
[5]White, C. T.: The weeds of Queensland. Austral. Queensland Agr. Jour., xx (1923), pp. 236-239.
[6]Anonymous: Poisonous properties of Sunn Hemp (*Crotalaria juncea*). Rhodesia Agr. Jour., xvii (1920), p. 497.

Ten Most Intelligent Animals

To settle once and for all the age-old debate as to which is the more intelligent, the horse or the dog, Dr. W. Reid Blair, director of the New York Zoölogical Park, has graded what he considers the ten most intelligent animals as follows:

1. Chimpanzee
2. Orang-utan
3. Elephant
4. Gorilla
5. Domestic dog
6. Beaver
7. Domestic horse
8. Sea lion
9. Bear
10. Cat

THE NATURE OF CANNIBALISM OCCURRING AMONG ADULT DOMESTIC FOWLS*

By C. H. WEAVER, *Poultry Pathology Laboratory*
Health of Animals Branch, and
S. BIRD, *Experimental Farms Branch*
Federal Department of Agriculture, Ottawa, Canada

Cannibalism is a term which by common usage has become descriptive for a condition occurring in poultry wherein birds are attacked by their pen-mates, are killed, and at times the body of the victim is partially consumed by the subjects of the attacking party.

Some question has arisen as to the suitability of the name but under the definition of "murderous cruelty" the designation, "cannibalism," is quite fitting even to those cases where there is an absence of flesh consumption on the part of the victors in the fracas. This fact will be more apparent when the following evidence is examined.

Examination of the available literature fails to show evidence of any attempts having been made to explain scientifically the cause of this phenomenal act, and it is surprising that definite studies have been neglected considering the ascending frequency of the trouble in recent years. It is of very great economic importance at the present time.

POULTRYMEN'S CONCEPTION OF CANNIBALISM

In the absence of definite knowledge as to the cause of cannibalism, it is to be expected that varied ideas would exist as to the nature of the trouble. Probably the most commonly advanced is that it is merely a vicious habit, developed in one or more individuals and possibly spread therefrom to other members of the flock. Based on this assumption, it is further believed that the attacked birds previously had suffered a prolapsus of the cloaca, which marks the subjects as victims of the attackers. Again it is thought that individuals laying upon the floor, instead of in the nests, are then exposed to attack while the vent is flaccid and relaxed.

Main emphasis, however, is placed upon the vicious habit aspect of the belief, looking to the attacking fowls as constituting the source of trouble, the attacked subjects being merely innocent victims.

*Received for publication, March 12, 1934.

Attention has been drawn to the seeming occurrence of cannibalism coincidental with the use of cod-liver oil as a corrective food ingredient.

NATURE OF CANNIBALISM

The cannibalistic attacks occur mainly while the attendant is absent and the fowls are alone by themselves. Should a person appear while an attack is in progress, it generally ceases, except in case the subject has been severely mutilated, when the onslaught may be pursued to its completion. Hence upon entering the quarters the attendant finds a dead fowl upon the floor, or an individual in a prostrate and dying condition, in either case partially disemboweled and very often the frenzied fowls running about, tearing to pieces and devouring the tissues torn from the body of the victim. With the excited and crazed condition of the flock occasioned by the melee and the taste of fresh flesh in their mouths, the birds do not immediately settle down to their usual complacent state of life but individuals may be seen passing among the pen-mates picking at the exposed parts of their bodies.

These scenes make a profound impression on the mind of the attendant and it is to be expected that spectacular rather than essential evidence should be carried away. His ire is naturally aroused against the active killers, and still more so if an occasional fowl with a good production record becomes the victim of a killing. In such cases it is quite conceivable that undue emphasis is placed on past performances to the obscuring of present conditions in existence in these as well as with the majority of the subjects.

INCIDENCE OF CANNIBALISM

In conducting autopsies on the cadavers coming from the flock of the Poultry Division, it has been made possible to compile a list which gives a fair indication of the incidence of the malady. These figures cover 3,664 cases, spread over a nine-year period, with a mortality from cannibalism of 440 subjects, which works out at an annual mean of 12 per cent of all fatalities. The highly important feature, however, is not the annual average but that in the first year of the series no deaths were recorded to cannibalism, while in the last year the listing attributable to this cause reached the enormous figure of over 25 per cent of all mortality. (See table I.) It was only in the fifth year that the trouble appeared in serious proportions, amounting to slightly less than 5.5 per cent of the flock. The following year, sixth in the series, it reached over 18.5 per cent. Any available evidence in

TABLE I—*Cannibalism incidence—Single-Comb White Leghorns.*

YEAR	OCT.	NOV.	DEC.	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	BIRDS	DEATHS	
															No.	%
H	–	–	–	–	–	–	–	–	–	–	–	–	–	462	0	.....
I	–	1	–	–	–	–	–	1	1	–	–	–	–	240	3	1.25
J	–	–	–	–	–	4	3	–	–	–	–	–	–	302	7	2.31
K	–	1	2	–	–	–	1	–	–	–	–	–	–	325	4	1.20
L	–	1	1	–	–	2	3	2	1	–	2	1	–	240	13	5.41
M	–	5	17	3	7	10	22	11	13	15	4	3	–	592	110	18.58
N	–	13	6	20	27	18	16	16	10	1	1	–	–	600	128	21.33
O	1	14	6	1	2	13	14	21	6	2	3	–	–	546	83	15.20
P	7	17	8	5	6	12	16	7	4	3	3	4	–	357	92	25.62
														3,664	440	12.00

explanation of this sudden rise is either too indefinite or insufficiently controlled to be of real significance.

CALENDAR OF SEASONAL DISTRIBUTION

During the last four years, deaths were occasioned from the trouble almost as soon as the fowls were confined in their permanent quarters. The mean monthly mortalities were:

Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.
2	12	9	7	11	13	17	14	8	5	3	2

This curve presents a sharp rise to a peak in the second month, followed by a lower plateau, with a second peak being built up during the sixth, seventh and eighth months. From thence the losses tail off quite sharply to the end of the year.

These figures establish the apparently normal manner of the occurrence of cannibalism. Because of the nature of the distribution of the deaths, which upon first consideration may appear as complicating the picture to its more difficult understanding, it in reality suggests avenues for consideration which may prove productive in arriving at the actual disease causes back of the abnormality.

RESPONSIBILITY FOR THE DEATHS

As these fowls were constantly trap-nested, egg-production can be made to serve a very useful purpose in obtaining a large part of the desired information concerning the placing of the responsibility for cannibalism. Accordingly, the production data of the fowls of the last, the "P" year (table I) have been put together as two groups: (a) the presumably normal fowls, those living the entire year, and (b) the birds which were killed by cannibalism. The production values were computed on a bird-day basis and are shown in tables II and III.

TABLE II—*Production values of normal and cannibalized birds.*

GROUP	BIRDS	MEAN NUMBER DAYS ALIVE	MEAN PRODUCTION (%)
Normal	109	366.0	44.4
Cannibalized	92	154.3	26.4

According to these statistics the cannibalized fowls decidedly constituted the poorer group. It has, however, been suggested that these fowls were in reality superior specimens, their excel-

TABLE III—*Monthly data on production values, expressed in percentages.*

GROUP	OCT.	NOV.	DEC.	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.
Normal	35.18	59.02	39.03	32.84	47.42	52.73	61.59	59.12	50.51	40.45	29.23	16.39
Cannibalized	19.66	35.64	25.68	25.50	24.24	30.47	29.77	24.26	20.58	15.68	11.23	.....

lence failing of recognition through the number of floor eggs produced by them, hence meeting their fate while thus exposed to attack.

The first part of this belief may easily be disproved. A calculation will show that the 92 cannibalized fowls, in order to compare in production with the normal fowls, would have had to produce in addition to the eggs recorded to them an average of 27.8 floor eggs, or a total of 2,558 eggs. By actual count, the whole flock of 305 birds was credited with a total of 632 floor eggs produced throughout the full twelve months. Even supposing the hypothesis was allowed that all of these eggs were produced by the 92 cannibalized birds, this would work out at 6.9 eggs each, or only 30.8 per cent mean production as against 44.4 per cent for the normal birds. This reflects to some extent against the second part of the belief.

Table IV, presenting the monthly incidences of cannibalism and of floor eggs, while showing an apparent similarity in the two curves, in reality argues against the general applicability of this belief.

TABLE IV—*Monthly incidence of cannibalism and floor eggs.*

	OCT.	NOV.	DEC.	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.
Incidence of cannibalism..	7	17	8	5	6	12	16	7	4	3	3	4
Floor eggs...	50	161	44	94	48	128	99	6	2	0	0	0

$$X^2 = 114 \quad n = 12 \quad p = 0$$

The X^2 measure for goodness of fit gives a "p" of 0, and it is therefore clear that one or more factors have intervened to produce a higher degree of dissimilarity in the distributions of these two curves than could possibly occur by chance, and the two sets of phenomena, for which they stand, are therefore not

related as uncomplicated cause and effect. Hence, the deduction may be drawn, that even though instances of cannibalism may from time to time result from the act of production being performed outside of the nests, they must have occurred so rarely that they cannot serve as a general or even a significant explanation of the trouble.

The other believed cause of cannibalism, namely, prolapsus, likewise must be given due consideration. Prolapsus of the oviduct is of itself a phenomenon not observed with any high degree of frequency, and should therefore seem to be of rather small importance. Further, no bird was reported killed the day she laid her last egg, and only ten instances were recorded as occurring within a week of this event. No bird, therefore, was killed immediately after prolapsus occurred, while the membranes were still invitingly exposed to attack in a moist and engorged condition; consequently prolapsus must have been of rather longer standing than would be very well conceivable in any but a decently managed flock, if this abnormality should have become a significant cause of cannibalism. However, it might be argued that production of floor eggs might in every instance have intervened between the last recorded egg and the occurrence of prolapsus, but in view of the rather small numbers of floor eggs recorded, coincidences of this nature could not have occurred with a frequency sufficiently high to make them of any significance.

This brings us to the most commonly accepted belief in the cause of cannibalism, that of a vicious habit on the part of the attacking fowls. If it were just that, then the belief presupposes an abnormality of the attacking fowls and normalcy among the others. In other words, being a habit, it would be peculiar to the habit-forming individuals. It is found, however, that the cannibalized fowls comprised a group which, on the average, were decidedly abnormal; many of them entirely without production, the remainder usually out of production when they were killed—possibly many of them sick at the time—and the majority of those which did produce eggs did so at a much slower mean rate than that of the normal fowls. Thus it would appear more logical to accept the subnormal state of the cannibalized fowls as the real cause of cannibalism. That is, the weaklings or those which have broken down under the existing conditions are killed off through a process of natural selection; a thing constantly going on in nature—the survival of the fittest.

The last of the suggestions set forth in explanation of cannibalism is its coincidental occurrence with the feeding of cod-

liver oil. While the practice of vitamin feeding to fowls has undoubtedly caused a marked increase in laying ability it is not believed to be supported by fact, that this can be, at best, more than a secondary cause (*vide supra*). It has been shown by Norris and Church[1] that cod-liver oil may contain substances which are toxic to fowls. Nevertheless, there is seemingly nothing to prove that this is in any way connected with cannibalism.

Before continuing further with the subject of the abnormality of the cannibalized group, previous to their being killed, let us first observe a naturally occurring variation.

A Breed Variation

The incidence of cannibalism is found to differ materially between the two breeds of fowls, Single-Comb White Leghorns and Barred Plymouth Rocks. According to figures previously reported,[2] in a population of 4,000 Leghorns and 1,810 Rocks there were cannibalized 14.38 per cent and 3.30 per cent respectively. These differences are too great in amount and occur with too definite regularity to make them a possible chance happening.

A possible explanation of this breed difference in incidence may be found in certain fairly well-defined breed characteristics. Knowing that the fowls which succumbed to cannibalism were mainly out of production at the time of their killing, attention was then directed to the manner in which the fowls of the respective breeds break in production.

Production pauses must in the main be looked upon as periods of recuperation, necessitated through the production output having exceeded the consumption of nourishment. Most fowls will keep up their output more or less intermittently while living partly on their body reserves, that is, while losing body weight. Those that in so doing lose the heaviest percentage of their body weight will naturally suffer the most when the break does occur, which it inevitably will, sooner or later. No doubt, therefore, the difference in body weight in the two breeds plays an important rôle in the incidence of cannibalism. The Leghorns, with an average body weight of some 1,700 grams, will suffer more from a normal weight loss than will the approximately 1,000-gram heavier Rocks.

Added to this comes the consideration that, during the time of active productivity, fowls are quite aggressive in keeping with the state commonly existing in the animal kingdom during stress periods in the reproduction of the species. This aggressiveness, however, generally is lost when a break occurs in production,

provided the birds do not at these times follow on to the natural sequence in events and go broody, in which case the aggressiveness is intensified.

Leghorns, as they are seen today, very seldom go broody, and a specimen of this breed suffering from a heavy loss in body weight and a consequent break in production will therefore mope about the pen, an easy prey to its aggressive pen-mates. Rocks, on the other hand, although not so prone to broodiness as in days gone by, nevertheless do go broody and as a breed retain much more of the broody urge than do the Leghorns. At the same time they suffer less from the comparatively slighter loss in body weight and, therefore, under most circumstances retain much more of the natural aggressiveness.

THE CAUSES OF CANNIBALISM

Since the evidence shows that the cannibalized fowls comprise a group of subnormal subjects, it becomes necessary to pursue the examination of them in an effort to determine the nature of the factors responsible for their condition and resulting in their killing.

The monthly percentage of production and mortality for this group through the "P" year are presented in table V. These two curves are found to be bimodal in character. This means either that in each curve a single factor has operated to give effect in the first, made to repeat its effect in the second, or else that two causes have contributed to the total results. It is believed that the bimodal effect in the production curve may be accepted, without further argument, as being caused by two factors. Presumably these are the accelerating urge to reproduce and the inhibitory

TABLE V—*Monthly production and mortality percentages for cannibalized fowls.*

MONTH	PRODUCTION (%)	MORTALITY (%)
October	19.66	8.0
November	35.64	19.3
December	25.68	9.1
January	25.50	5.7
February	24.24	6.8
March	30.47	13.6
April	29.77	18.2
May	24.26	8.0
June	20.58	4.5
July	15.68	3.4
August	11.23	3.4

highly adverse midwinter conditions during the months of December, January and February.

As the modes of these two curves coincide so nearly as to time, mortality might prove to be but a reflex of production if the general fit should prove to be a good one.

The measures for goodness of fit were found to be:

$$X^2 = 8.64 \quad n = 11 \quad p = .65$$

This means that a fit as good as the one observed would occur, by chance, twice out of every three trials, and mortality from cannibalism may therefore be taken to be closely correlated to production. This conclusion fits very well the conception of aggressiveness as being a marked characteristic of laying fowls. However, it was found that the mean number of days the cannibalized group of birds were out of production immediately preceding the actual killing was 46.08 ± 3.695, with a standard deviation of 35.25 ± 2.606 days, the coefficient of variability being 76.5 per cent. This means that there were always present in the pens subnormal individuals, that, at the time, when they fell prey to the natural aggressiveness of their normal pen-mates, might have been out of production for a few days only or for four to five months. In other words, the incidence of cannibalism is not, as might be deduced from table V, correlated to the production of the slaughtered birds themselves at the actual time of killing, as they, generally speaking, had been out of production, at the time when the fatalities occurred, for about six weeks. This incidence, however, does correlate very intimately with the productivity and attendant aggressiveness of the birds that were normal or near normal at the time when the killings took place. The closeness of fit between the production curves of the normal and low-producing, later on cannibalized, birds may be appreciated from table III. Presumably it did not make much difference whether some of the killers later became subnormal themselves and in their turn were killed off after a shorter or longer term of lingering in the pen.

The question then remains how a disproportionate number of the cannibalized birds, great enough to make 26 per cent, could be or become subnormal to such an extent as to lay themselves open to attack as early in the year as during the first two months.

To clear up this point, the distributions of the mortality groups were constructed as shown in tables VI and VII.

TABLE VI—*Age groups (group intervals, 40 days).*

BIRDS DEAD FROM	DAYS										TOTALS
	40	80	120	160	200	240	280	320	360	400	
Cannibalism	16	16	6	8	21	11	4	5	5	....	92
Avitaminosis	....	....	....	2	1	0	2	3	3	....	11
Parasitism	6	8	4	2	1	....	....	....	1	....	22
Respiratory	1	5	7	15	1	1	2	....	....	....	32
Peritonitis	1	0	1	2	1	6	2	3	1	3	20
Reproductive	2	1	1	0	5	2	1	5	2	....	19
Totals	26	30	19	29	30	20	11	16	12	3	196

TABLE VII—*Production groups (group intervals, 20 eggs).*

BIRDS DEAD FROM	EGGS											TOTALS
	20	40	60	80	100	120	140	160	180	200	220	
Cannibalism	43	8	16	8	6	4	3	1	1	1	1	92
Avitaminosis	....	2	1	1	1	1	4	1	....	....	....	11
Parasitism	11	7	2	1	0	1	....	....	....	....	....	22
Respiratory	7	12	4	5	3	1	....	....	....	....	....	32
Peritonitis	4	3	2	3	3	3	1	1	....	....	....	20
Reproductive	4	3	6	1	3	0	1	0	1	....	....	19
Totals	69	35	31	19	16	10	9	3	2	1	1	196

The three groups, avitaminosis, peritonitis and reproductive, are made up of birds either too few in number or too indefinite in character for normal distributions to occur. The respiratory diseases (items 2, 3, and 6, table VIII) as well as parasitism, on the other hand, are quite definite groups and they form normal and fairly regular distributions, without any indication of bimodal arrangement. The significant fact, however, is that the total mortality distribution, a composite of all the smaller ones, being expressive of the occurrences in a mixed population is bimodal or trimodal.

Here again the parasitism and respiratory groups are fairly normal, the other groups being spread out, probably because the classification had to cover a variety of conditions. From the fact that parasitism is mainly responsible for the early mode of the total mortality distribution, exclusive of cannibalism, it is therefore believed to be indicated that the majority of those cannibalized birds producing below 20 eggs and living less than 80 days

TABLE VIII—*Mortality—Single-Comb White Leghorns.*

ITEM	DISEASE	OCT.	NOV.	DEC.	JAN.	FEB.	MAR.	APRIL	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	MISC.	BIRDS	RELATIVE PERCENTAGE	ABSOLUTE PERCENTAGE
1	Intestinal parasitism	4	2	7	3	3	—	1	—	1	—	—	1	—	—	22	8.87	6.16
2	Roup and chicken-pox	—	—	1	1	1	—	—	—	—	—	—	—	—	—	3	1.20	0.84
3	Roup only	—	1	—	2	3	—	—	1	—	1	—	—	—	—	8	3.22	2.24
4	Pox only	—	1	2	3	9	3	—	—	1	—	—	—	—	—	19	7.66	5.32
5	Laryngotracheitis	—	1	—	—	—	1	—	—	—	—	—	—	—	—	2	0.80	0.56
6	Pullorum disease	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
7	Tuberculosis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
8	Pericarditis	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	0.40	0.28
9	Vent gleet	—	—	1	—	—	—	—	2	1	1	—	—	—	—	5	2.01	1.40
10	Peritonitis	1	—	—	1	2	—	1	6	2	1	3	1	2	—	20	8.06	5.60
11	Reproductive	1	1	1	1	—	3	3	2	—	2	3	2	—	—	19	7.66	5.32
12	Avitaminosis	—	—	—	—	—	3	—	—	—	2	5	1	—	—	11	4.43	3.08
13	Cannibalism	7	17	8	5	6	12	16	7	4	3	3	4	—	—	92	37.09	25.77
14	Hemorrhage	—	—	—	—	—	—	1	1	—	—	—	—	—	—	2	0.80	0.56
15	Visceral gout	—	—	—	—	—	1	—	—	—	—	—	1	—	—	2	0.80	0.56
16	Digestive and liver troubles	—	—	1	2	—	—	—	—	—	1	—	—	1	—	5	2.01	1.40
17	Tumor	—	1	—	—	—	1	—	—	1	1	1	—	1	—	6	2.41	1.68
18	Leucosis	2	—	—	—	—	—	—	1	—	1	1	1	—	—	6	2.41	1.68
19	Paralysis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
20	Heat prostration	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
21	Undetermined	—	1	—	—	—	—	—	1	1	1	—	2	1	—	7	2.84	1.96
22	Miscellaneous	—	—	—	—	—	2	—	—	1	—	—	—	—	15*	18	7.25	5.04
	Totals	15	25	21	18	24	26	22	21	13	14	16	13	5	5	248		
	Relative percentage	6.04	10.08	8.46	7.25	9.67	10.48	8.87	8.46	5.24	5.64	6.42	5.24	2.01	6.04			
	Birds adjusted for percentage calculation	357	342	317	296	278	254	228	206	185	172	158	142	129	124			
	Absolute percentage	4.20	7.30	6.62	6.08	8.63	10.23	9.64	10.19	7.02	8.13	10.12	9.15	3.87	12.09		69.46	

*Birds that had died or were killed by the management and were not submitted for autopsy.

were set upon by the other birds in the pen, on account of the weakened condition that parasitism had induced in them.

If, in accordance with this view, some 38 birds are eliminated from the cannibalized group producing below 20 eggs each, that is, arbitrarily eliminating all birds producing less than 10 per cent, then the distribution of the age groups would be as shown in table IX.

TABLE IX—*Distribution of age groups after eliminating all birds producing less than 10 per cent.*

BIRDS DEAD FROM	DAYS									TOTALS
	40	80	120	160	200	240	280	320	360	
Cannibalism	2	5	3	7	16	9	3	4	5	54
Cannibalism (and supposed parasitism)	14	11	3	1	5	2	1	1	....	38
Parasitism	6	8	4	2	1	....	....	....	....	21

These three curves are fairly normal. The pure cannibalized group indicates a positive correlation to the spring production peak. The two other groups indicate that parasitized birds have 35 per cent chances of dying a natural death and 65 per cent chances of being killed by their pen-mates. They also indicate that if the effects of parasitism could be eliminated, practically all of the early winter peak, found in this instance, would disappear. This would mean that the cannibalism problem would be so much less complicated to deal with, and may suggest a further avenue of attack for clearing up of the whole complex problem of subnormality and cannibalism.

A comparison of the normal birds with the group of 54 cannibalized birds shown in table IX is shown in table X.

The percentage production shows a statistically very highly significant difference, as shown in table XI.

This means that the distribution curves describing these two populations are very much of the same order. The differences

TABLE X—*Comparison of production in normal and cannibalized birds.*

GROUP	BIRDS	MEAN NUMBER DAYS ALIVE	MEAN PRODUCTION (%)
Normal	109	366.0	44.4
Cannibalized	54	189.6	35.2

TABLE XI—*Percentage production differences in two groups.*

GROUP	BIRDS	MEAN PRODUCTION (%)	STANDARD DEVIATION	COEFFICIENT OF VARIABILITY
Normal	109	44.4 ± 1.64	16.91 ± 1.15	38.12 ± 2.93
Cannibalized	54	35.2 ± 2.25	16.35 ± 1.59	46.51 ± 5.42
		DIFFERENCES		PROBABILITY
Mean productions (%)		$9.2 \pm 2.780 \frac{9.2}{2.78} = 3.305$		0.0012
Standard deviations		$1.56 \pm 1.959 \frac{1.56}{1.959} = 0.286$		0.78
Coefficients of variability		$8.39 \pm 6.168 \frac{8.39}{6.168} = 1.36$		0.18

found between standard deviations and coefficients of variability would occur as a matter of chance 78 and 18 times in a hundred, respectively. On the other hand, the difference established between the mean per cent productions would occur as a matter of chance only 12 times in 10,000. This difference, therefore, is very highly significant and probably is due to the same weakened conditions in the cannibalized birds that brought about this fatality.

It might be argued that the production percentage of the normal group of birds has been calculated on a 366-day period, as against 190 days for the cannibalized group. This might conceivably make the production percentage not comparable. It was found, however, that there was no correlation at all between production percentage and length of life and that the production for the normal birds over the first 190 days of their pen life was 50.6 per cent. As this but widens the difference between the two groups, as does the use of the 26.4 per cent production figure for the original group of 92 cannibalized birds, there can be no doubt of the significant difference established, no matter how the figures are treated.

These fowls suffered a severe attack of coccidiosis during the latter part of their range period, that is, but a short time before they were moved into the houses whence these figures were com-

piled. Considerable mortality was occasioned at the time and many remaining fowls were left in a weakened condition as a result of the parasitism. This likely played an important rôle in the subsequent eventualities.

DISCUSSION

The compilations herein presented were collected upon a single poultry plant and are not necessarily representative of flocks in general, though it does seem probable that they are not greatly dissimilar from what would obtain with other flocks when conducted under comparable conditions. There are no means at hand to determine whether or not the flock in question is an exceptional case in general or in detail.

A very decided increase occurred in recent years which is believed to be other than an isolated case. There may be a slight exaggeration in the rise, as shown through a practice of flock culling in operation in all except the last year. Birds so removed, being culls, would be more susceptible to cannibalism than those left behind. This practice precludes a detailed study except for the one year as has been done.

No attempt has been made to record all the divergent views regarding the cause of cannibalism, but merely to take into consideration those most frequently brought to our attention. Since, in these instances, the ones considered are obviously unfounded in fact, there are no reasons for believing that others would be less erroneous.

The evidence leaves little doubt of the cannibalism having been in the nature of a complex as to cause. A large part of it undoubtedly was induced by the subnormal physical condition of the killed subjects. In such a case this natural culling should tend to an improvement of the health status of the flock in subsequent generations. It has been shown, however, that the opposite prevails in that an ascending mortality is recorded. A most likely explanation of this reversal of an anticipated result is that certain factors, environmental or otherwise, resulting in the undermining of the health of the fowls leading to their killing, were so overwhelmingly greater influences than the beneficial results made possible by the culling as to render them quite negligible.

The important recording is the fact that the production of the cannibalized fowls as a group was less than the normal fowls with the calculations made on a bird-day basis. This may be taken

as being indicative of a subnormal state of the individuals comprising the group.

REFERENCES

[1]Norris, E. R., and Church, A. E.: The toxic effects of fish liver oils and the action of vitamin B. Jour. Biol. Chem., lxxxix (1930).

[2]Weaver, C. H.: A study of the causes of mortality in groups of the Single-Comb White Leghorn and Barred Plymouth Rock breeds of fowl; including a comparison between production and mortality. Proc. 4th World's Poultry Congress, London, England, 1930.

Zoological Building Dedicated

The new Zoölogical Building at the Beltsville Research Center, Beltsville, Md., was dedicated on October 20, 1934, in the presence of more than 100 guests prominent in the fields of zoölogy and allied sciences. Dr. Maurice C. Hall, chief of the Zoölogical Division of the U. S. Bureau of Animal Industry, presided and several guests gave short talks. The speakers included: Dr. Cooper Curtice, who was the first chief of the Division; Dr. Charles Wardell Stiles, who also served as chief of the Division; and Dr. Albert Hassall, who until his recent retirement was assistant chief. They discussed the early work of the Bureau in research and procedures for the eradication and control of animal parasites, and stressed the possibilities that the new building and equipment provide for additional investigations.

Appearing on the program also were: Dr. Henry B. Ward, permanent secretary of the American Association for the Advancement of Science; Dr. W. E. Cotton, superintendent of the Bureau of Animal Industry Experiment Station, at Bethesda, Md.; Dr. Lawrence A. Avery, superintendent of the new laboratory; and Earl C. Butterfield, newly appointed superintendent of the Beltsville Research Center. Other guests represented Johns Hopkins University, the Helminthological Society of Washington, the Smithsonian Institution, and various branches of the Department of Agriculture.

The building cost $50,000 and contains 31 rooms. It will be used for experimental work in the control and eradication of parasites. Several other buildings accommodate small animals such as dogs, cats, guinea pigs, rabbits, chickens, pigs and others. The special equipment includes a variety of apparatus, a post-mortem room, refrigerator room, incinerator and library. The lots and pens outside these buildings are separated by double fences and ditches to prevent contamination from other pens.

"Don't you think that new popular song is good?"
"Oh, fair to maudlin."

THE RELATION OF ABORTION OR BANG'S DISEASE TO MASTITIS*

By CECIL ELDER, *Columbia, Mo.*

Department of Veterinary Science, University of Missouri

The problem of contagious abortion or Bang's disease and that of mastitis are both of major importance and naturally we should be interested in the relationship between the two. When your secretary so kindly invited me to appear on your program, I was somewhat hesitant about accepting the invitation, due to the fact that our work on the problem has been so limited.

Knowing that you should be interested in the work that has already been done in this connection, I thought it advisable to review the literature for you. This review will necessarily be brief, as there has not been a great deal published upon this particular phase of the problem. A large number of references in the literature can be cited regarding the economic losses from Bang's disease, especially those losses due to low milk-production in positive reacting animals, as compared with negative animals handled and fed in the same way.

Several of the experiment stations in the United States have worked on this problem and estimated the cost, in dollars and cents, of the losses that can be actually attributed to Bang's disease infection. Very few of them make any reference to the relation of Bang's disease to mastitis and the part that such mastitis plays in the lowered production. It is a well-demonstrated and proven fact that many Bang's disease-infected animals eliminate *Brucella abortus* in the milk from their udders, but we do not have much definite information as to what part this udder infection with *Br. abortus* plays in relation to mastitis. Mastitis is often divided into the non-infectious and infectious types. It is the opinion of some that with the non-infectious type of mastitis little damage is done to the udder, and that the inflammation of the parenchymatous tissue may last only a day or two, while in the infectious type of mastitis, which is generally considered to be due to streptococci and other pathogens, great damage, and probably in most cases permanent injury, is done to the udder.

The question which I should like to discuss here is what part does *Br. abortus* play in the causation of mastitis? Cooledge,[1] in his studies upon the bacterial flora of milk with high cell-counts,

*Contribution from the Department of Veterinary Science, Missouri Agricultural Experiment Station, Journal Series No. 389. Presented at the joint meeting of the Saint Louis District Veterinary Medical Association and the Illmo Veterinary Medical Association, Collinsville, Ill., March 29, 1934.

stated that *Br. abortus* does not cause microscopic lesions in the cow's udder, but he thinks that it may be the cause of high cell-counts in milk because *Br. abortus* can produce such great changes in other tissues. He found that milk from actively *Br. abortus*-infected udders had a cell-count nearly five times as high as that of the apparently normal average, which he considered to be 868,000. Udders artificially infected with *Br. abortus* were quick to show high cell-counts.

Tweed[2] later verified Cooledge's work, but I believe found that although the cell-count of the milk was increased, it was not increased to so great an extent as indicated by Cooledge. Tweed is of the opinion the most common pathogenic organism that causes alterations in the glandular tissue of the udder is the streptococcus. He states that "*Brucella abortus* has not been proved to be a cause of any gross or microscopical lesions of the cow's udder." At the same time it is interesting to note that *Br. abortus* can produce pathological changes in the tissues of certain experiment animals. Tweed reported that the increased cell-content was due to increased leucocytes, and not to epithelial cells.

Friedemann, about 1921, according to Giltner, Huddleson and Tweed,[3] undertook to determine the pathological changes that take place in a Brucella-infected udder. He worked under very carefully controlled conditions with only one positive udder and one negative control. His results were negative as far as histological changes are concerned. In other words, his findings in one udder did not differ from the findings in the other. He was of the opinion that *Br. abortus* udder infection is not detectable by either macroscopic or microscopic desquamative or suppurative processes.

Giltner, Huddleson and Tweed,[3] in a report in 1922, state that "we do not know whether udder infection is primary or secondary, but probably cases of both types occur." They further state that "the changes produced in the udder seem to have been sought by Schroeder and Cotton at autopsies, with negative results." They also thought that another factor throwing doubt on the possibility of finding lesions in so-called infected udders was the very small number of *Br. abortus* organisms found in the milk. In some of their work the number isolated averaged 80 per cc of milk.

Simms and others[4] state that in their work with the Oregon herd the reacting cows had more garget. In 44 reacting cows there were 31 cases of garget during the three-year period, while

during the same time there were only two cases of garget in 27 abortion-free cows.

A few men have suggested the hypothesis that *Br. abortus* is the primary cause of mastitis, and paves the way for the streptococci and other bacteria which may be isolated as secondary factors. In an attempt to prove or disprove this, Carpenter[5] injected a series of guinea pigs with milk samples which came from mastitis cases. He injected guinea pigs with 77 such samples and, after a period of six to eight weeks following injection, the pigs were killed and cultured and blood tests made. Three of the pigs so injected were found to be infected with *Br. abortus*. The blood serum from these pigs had a titre of 1:960 and the pigs showed typical lesions, from which pure cultures of *Br. abortus* were isolated. These three pigs represented only 3.89 per cent of the samples. It is interesting to note that these samples came from herds with considerable Bang's disease infection, and in which there were many high-titre animals. The same number of milk samples as were used here will show a much higher percentage of *Br. abortus* than the percentage of samples from the inflamed udders used.

Carpenter[5] was of the opinion that the alpha hemolytic type of streptococcus (or the non-hemolytic type of Jones) is responsible for the majority of losses caused by mastitis. Other workers are of the opinion that streptococci are by far the most frequent invaders of the udder.

Madsen and Hendricks,[6] of Utah, in their work with Bang's abortion disease, make the following statement:

> Breeding efficiency is lower and such cows are more subject to udder infections.

In the work at Wisconsin, in connection with the relation of nutrition to Bang's disease, Hart, Hadley and Humphrey[7] found that several cows on experiment developed mastitis, but those workers were of the opinion that this mastitis was no different from that in the average herd, and in no case could it be attributed to infection with *Br. abortus*.

Going back again in the literature, we find that Theobald Smith *et al.*[8] thought that the invasion of the udder by *Br. abortus* means its presence and multiplication in the milk in the acini and ducts, and not in the connective tissue. In other words, the residual milk in the acini and ducts, rather than the udder tissue, appears to be the seat of multiplication.

Buck[9] states:

> Unlike numerous varieties of germs, the abortion bacilli, when present, do not appear to produce harmful results in the udder. The

multiplication of the germs in this organ seems to be merely a provision of nature for the perpetuation of the infection during those intervals when conditions are unfavorable for growth of the germs in the uterus.

Runnells and Huddleson,[10] reporting on four cases, both bacteriologically and histologically, found three of the four showed *Br. abortus* bacteriologically. Microscopic lesions thought to be due to *Br. abortus* were found in the gland tissue. The lesions found were an increase of interstitial tissue and foci of marked cellular infiltration—cells consisting of lymphocytes, polymorphonuclear leucocytes, eosinophils, plasma cells and polyblasts. No gross lesions were noted.

Sholl and Torrey[11] studied the milk and udders from many cows and found that interstitial mastitis appeared to be the predominating type associated with *Br. abortus* infection in the udder. In cows with positive reactions where *Br. abortus* was isolated there was a predominance of interstitial mastitis. In negative cows where streptococci were isolated there was a predominance of exudative mastitis. In their article, however, they describe finding interstitial mastitis also in such cases. It is interesting to note that they were seldom able to isolate *Br. abortus* when they found streptococci. They concluded that "*Brucella abortus* produces definite changes in the udder which may be quite extensive and may lower the efficiency of the organ considerably."

In 1932, Birch,[12] of New York, reported that in heifers with *Br. abortus* infection there develops a mild and more or less transient mastitis involving one or more quarters of the udder.

Mitchell[13] was of the opinion that pathological lesions resulting from the presence of *Br. abortus* are few indeed. In fact, excepting pathological changes brought about in the placentae of pregnant animals, scarcely a lesion of note has been found. He was of the opinion that there is no clinical evidence of Brucella infection in the udder.

From the above review of the literature it would appear to me that the gross lesions in udders of cows showing infection with *Br. abortus* are not very significant. It is true that some have found in such animals an interstitial mastitis, which probably was the result of the Brucella infection. However, we very often find interstitial changes in cases of mastitis due to other organisms, such as the streptococci and other pathogens. In my opinion it is extremely difficult to differentiate between these two conditions, and to determine definitely whether or not this inter-

stitial mastitis is due to *Br. abortus* or to some of the streptococcus group. It has been found practically impossible to recognize *Br. abortus* in histological sections. At any rate, *Br. abortus* does not produce gross pathological lesions, and it is difficult to prove that cases of mastitis are due to this infection. For some time I have been interested in its possible relationship, but at the present time our work on the problem has not gone far enough to draw definite conclusions. In this connection, you may be interested in some of the observations which we have made at the University of Missouri.

As you know, Dr. J. W. Connaway was one of the pioneers in the investigation of Bang's disease, and was one of the early workers in the use of the blood-test as a means of diagnosing this infection in cattle and swine. As a result of the early work started by him we have an excellent foundation on which to build and continue with our experimental work and studies of this disease.

In connection with this blood-testing program in our laboratories it has been our privilege to have an opportunity to work with several of the state institution herds of Missouri. I am glad to say that most of them are now accredited, or almost ready for accreditation as Bang's disease-free herds. While carrying on this blood-testing program we have had opportunity to make many observations upon these herds. At several of the state institutions the positive and negative animals were divided into separate units and were cared for by different sets of attendants, in separate barns, as in several cases patients in the institution were used to do the work. For all practical purposes it appears that the negative and positive herds received the same rations, same care, and both were housed in good barns. We noted in this connection that the herdsman always reported more trouble from mastitis or garget, as they call it, in the positive herd than in the negative herd. Typical of these reports is one in which the herdsman in charge of one of the herds reported that their veterinarian, working with the herdsman, had made a very careful, thorough check on mastitis in both the positive and negative herds at that institution. They found that they had had very little or no trouble in the negative herd, but that a number of cases of mastitis had occurred in the positive herd. It appears that only two or three of these were chronic cases. He further reports that he has found in his observation of the two herds that the highest reacting individuals to the agglutination test are more frequently the ones to develop mastitis.

In another large herd, according to figures furnished me, 43 animals out of the original 47 that reacted positively to the blood-test had mastitis in one or more of its various manifestations. In the negative herd at the same institution the first survey indicated only 22 out of 64 had reacted to the brom thymol blue test. The technician in a well-equipped laboratory had isolated both staphylococcus and streptococcus from animals that showed mastitis in this herd.

This would lead one to believe that infection with *Br. abortus* certainly had a direct relationship with mastitis. On the other hand, in herds which we know have been free from *Br. abortus* infection for several years we have observed very severe outbreaks of mastitis, in most cases due to streptococci, so we cannot conclude that the Bang's disease infection alone will necessarily have any effect upon the incidence of mastitis.

We have further had the opportunity to see and examine several of these positive animals when they were killed after they became unproductive for one reason or another. We have in no case found any gross pathological lesions in the udders of such animals. Unfortunately, our work on histological sections from these udders is not completed at the present time.

It has been of further interest to me to note that in our experiment animals, several of which we have artificially infected with *Br. abortus*, we have never noticed any clinical evidences of mastitis, with possibly one exception. Here, however, we should keep in mind that the animals which we have had on experiment were both of the beef and dairy types but none of them were high milk-producing animals. We would naturally expect less mastitis in such animals than we would in the higher producing group.

In conclusion, on a basis of the reports in the literature and the observations which we have made in our work with Bang's disease, I do not believe *Br. abortus* to be an important factor in the production of any gross lesions in the udder, but I do think *Br. abortus* infection may act as a predisposing cause and pave the way for the entrance of streptococci and other organisms which seem to be most important in the causation of bovine mastitis.

REFERENCES

[1]Cooledge, L. H.: Studies upon the bacterial flora of milk with high cellular counts. Mich. Agr. Exp. Sta. Tech. Bul. 41 (1918).

[2]Tweed, R. L.: The relation of high cellular counts to *Bact. abortus* infection of the udder. Mich. Agr. Bd. Rpt. (1924), pp. 872-896.

[3]Giltner, W., Huddleson, I. F., and Tweed, R. L.: The rôle of the udder and its secretion in bovine infectious abortion. Jour. A. V. M. A., lxii (1922), n. s. 15 (2), pp. 172-178.

[4]Simms, B. T., *et al.*: The eradication of infectious abortion from the dairy herd of Oregon State Agricultural College. Ore. Agr. Exp. Sta. Bul. 232 (1928).
[5]Carpenter, C. M.: The bacterial content of milk or inflammatory exudates from bovine mastitis. Jour. A. V. M. A., lxvii (1925), n. s. 20 (3), pp. 317-323.
[6]Madsen, D. E., and Hendricks, W. H.: Control and eradication of Bang's abortion disease. Utah Agr. Exp. Sta. Cir. 94 (1931).
[7]Hart, E. B., Hadley, F. B., and Humphrey, G. C.: The relation of nutrition to contagious cattle abortion. Wis. Agr. Exp. Sta. Res. Bul. 112 (1932).
[8]Smith, T., Orcutt, M. L., and Little, R. B.: The source of agglutinins in the milk of cows. Jour. Exp. Med., xxxvii (1923), p. 153.
[9]Buck, J. M.: Infectious abortion in cattle. U. S. Dept. of Agr. Farmers' Bul. 15-36 (1927).
[10]Runnells, R. A., and Huddleson, I. F.: The nature of *Bact. abortus* infection in the udder of the bovine. Corn. Vet., xv (1925), p. 376.
[11]Sholl, L. B., and Torrey, J. P.: A contribution to the bacteriology and pathology of the bovine udder. Mich. Agr. Exp. Sta. Tech. Bul. 110 (1931).
[12]Birch, R. R.: The nature and economic importance of Bang's disease. Rpt. N. Y. State Vet. Coll., Cornell Univ. (1932), pp. 163-170.
[13]Mitchell, C. A.: *Brucella abortus* infection of the udder. Rpt. Int. Asso. Dairy & Milk Inspectors (1931), pp. 231-241.

Phi Zeta Enters Ohio State

Delta Chapter of Phi Zeta, honorary veterinary fraternity, was installed at Ohio State University, April 3, 1934. Dr. R. R. Birch, of New York State Veterinary College, national president of Phi Zeta, conducted the installation ceremonies. Seven faculty members and eight students became charter members of the new chapter, with Dean O. V. Brumley, honorary member of Alpha Chapter, elected president for the coming year.

In installing the chapter, Dr. Birch traced the origin of honorary societies and gave a brief history of the beginnings of Phi Zeta. He also outlined the purposes which such societies serve. In presenting certificates to the newly elected members, Dr. Brumley expressed the satisfaction of the veterinary faculty at having a chapter of Phi Zeta at Ohio State and stated that each member individually had a responsibility in promoting the welfare and usefulness of the organization.

The Fraternity held its first regular election on May 14, and elected 19 students to membership. The new members included five graduate students, nine seniors and five juniors. Elections are based on scholarship and research.

W. A. H.

Benefit Ball for Royal College

H. R. H. The Duke of Gloucester is to be the patron of a benefit ball to be given January 4, 1935, to aid the rebuilding fund of the Royal Veterinary College, of London. Prominent on the committee that is planning the event is Professor Sir Frederick Hobday, principal of the College.

CALF LOSSES DUE TO IODIN DEFICIENCY*

By ROY H. MILLS, *Sacramento, Calif.*
Division of Animal Industry
California Department of Agriculture

Early in 1933, the Division of Animal Industry was called upon to investigate calf losses in the Hat Creek section of eastern Shasta County, in the northern part of California. Hat Creek has its source in the snows of Mount Lassen, the only active volcano in the United States, and flows its entire length through lava formation, emptying into Rising River.

During 1932, a veterinarian of the Division visited this valley to investigate premature calf losses. The results of blood samples taken from a herd of cattle and submitted to a laboratory for the agglutination test showed several animals positive to Bang's disease. Therefore, when a request for aid was received last year, arrangements were made to take blood samples from several herds in order to determine whether this disease was the cause of the trouble.

A range herd of Hereford cattle was visited first. The calves born in the fall arrived at practically full time, but showed evidence of thyroid enlargement when several months old. Many of the spring calves, dropped in the extremely cold weather of January, February and March, were premature, as shown by lack of incisor teeth, and also very weak and small. Many showed evidence of goiter and a few were hairless. Most of these calves were alive at birth and, if given good care for a week or more, survived, but were much smaller than normal. (See figure 1.) The cows were range animals and rather difficult to confine in order that the calves could be helped to suck their mothers. Between 40 and 45 died because of these insurmountable obstacles.

One calf was autopsied. The region of the thyroid was enlarged and the subcutaneous tissues in this area were infiltrated with a gelatinous substance. (See figure 2.) The neck muscles were very hemorrhagic and infiltrated with blood. The thyroids were about the size of a turkey egg and rather soft in consistency. The rest of the organs were normal.

Many of the cows that had given birth to weak, premature calves retained their fetal membranes. Several died as a result of metritis which subsequently developed. Eight animals that had given birth to premature calves, representing a cross-section

*Received for publication, April 13, 1934.

of the herd, were bled from the jugular. Agglutination tests proved these animals to be free from Bang's disease.

Similar trouble was being experienced in a dairy herd further up the creek, where it was discovered that the greatest calf loss occurred during the extremely cold weather. Many calves had shown goiter at birth and were very weak; some were hairless, and a number of the cows had retained the placenta. As this was a dairy herd under good management, it was possible to obtain a fairly satisfactory record, which is presented in table I.

FIG. 1. One-day-old calf. Could stand only a few seconds when placed on its feet. Assisted to nurse and made recovery.

Out of 16 cows in this herd on which we have breeding records, 12 retained the afterbirths. Nine of the 16 calves dropped either died or were dead at birth. Two cows were positive to the agglutination test for Bang's disease. The one on which we have a breeding history had delivered a normal calf; breeding history was not available for the other. The incidence of contagious abortion evidently was not responsible for the trouble in this herd. The greatest calf mortality occurred during February and March, when the weather was exceedingly inclement.

The breeding history of cow 2 for two years tends to show a cumulative effect of a prolonged deficiency of some nature, and

TABLE I—*No iodized salt supplied until about March 1, 1933.*

Cow	Bred	Calved	Time Calf Carried	Condition of Calf	Retained Afterbirth	Agglutination Test (3–24–33)
1	7–4–31	4–13–32	9 mo. 9 da.	Fully developed; weak; died	Yes	Not tested
2	8–23–31	6–1–32	9 mo. 8 da.	Fully developed; dead	No	Negative
	7–28–32	3–6–33	7 mo. 8 da.	Goiter; dead	Yes	Negative
3	——	12–13–32	——	Fully developed; dead	Yes	Negative
4	——	1–18–33	——	O. K.	Yes	Negative
5	11–26–31	9–8–32	9 mo. 8 da.	O. K.	No	Negative
6	11–3–31	8–8–32	9 mo. 5 da.	O. K.	No	Negative
7	——	——	——	——	——	Negative
8	——	3–9–33	——	Goiter; dead	Yes	Negative
9	——	3–7–33	——	Goiter; dead	Yes	Negative
10	——	3–7–33	——	Goiter; died	Yes	Negative
11	11–7–31	8–12–32	9 mo. 5 da.	O. K. (vealed)	No	Positive
12	——	12–28–32	——	Hairless; dead	Yes	Negative
13	——	12–17–32	——	O. K.	Yes	Negative
14	——	——	——	——	——	Negative
15	——	10–22–32	——	O. K.	No	Negative
16	——	——	——	——	——	Positive
17	——	1–13–33	——	Died 3rd week; scours	Yes	Negative
18	——	——	——	——	——	Negative
19	——	3–15–33	——	Goiter; dead	Yes	Negative
20	——	12–12–32	——	O. K.	Yes	Negative

indicates that premature births or abortions can occur from such a deficiency. It is unfortunate that the breeding dates of cows 8, 9, 10, 12 and 19 could not be secured, because we feel sure they would have proved that premature births are possible even in the absence of positive agglutination tests to Bang's disease.

FIG. 2. Stillborn calf showing enlargement of thyroid.

These investigations were superseded by a study of the condition in this section by Dr. George H. Hart, of the University of California, who recommended the feeding of iodized salt. A ranch was visited where this recommendation had been followed. About one year after the owner moved to this place, practically all calves born were very weak, and many showed goiter. The

TABLE II—*Iodized salt in bricks used.*

COW	BRED	CALVED	TIME CALF CARRIED	CONDITION OF CALF	RETAINED AFTERBIRTH	AGGLUTINATION TEST (3–24–33)
1	6–20–32	3–22–33	9 mo. 2 da.	Twins, both small; 1 died	No	Negative
2	3–15–32	12–29–32	——	O. K.	No	Negative
3	3–9–32	8–27–32	5 mo. 18 da.	Dead	Yes	Negative
4	4–18–32	12–29–32	8 mo. 11 da.	O. K. (vealed)	No	Negative
5	4–19–32	2–10–33	10 mo. 21 da.	O. K.	No	Negative
6	2–20–32	11–24–32	9 mo. 4 da.	O. K. (vealed)	No	Negative
7	9–3–32	2–17–33	5 mo. 14 da.	Weak; died	No	Negative
8	8–25–31	6–6–32	9 mo. 11 da.	Weak; died	No	Negative
9	5–23–32	——	——	——	——	Negative
10	4–2–32	1–14–33	9 mo. 12 da.	O. K.	No	Negative
11	7–15–32	——	——	——	——	Negative
12	9–1–32	6–2–33	9 mo. 1 da.	O. K.	No	Negative
13	12–18–32	——	——	O. K.	No	Negative
14	——	4–2–32	——	Dead; scours	No	Negative
15	——	——	——	O. K.	No	Negative
16	6–10–32	3–19–33	9 mo. 9 da.	O. K.	No	Negative

feeding of iodized salt to the cattle was then adopted, although there were occasional periods when the animals were without this preparation. At the time of our investigation, only a very small percentage of calves on this place were showing goiter. Table II gives a record of this herd.

Other owners along Hat Creek, both of beef and dairy cattle, were experiencing similar troubles. One beef cattle herd had the same difficulties when formerly located in the Hat Creek area, but after the cattle were moved to a place on Rising River, the cows retained their calves full time, gave birth to normal young, and

FIG. 3. Old dog showing a goiter the size of a pineapple.

did not retain the afterbirths. Inquiry was made as to whether similar experiences had been encountered along Rising River. Many cattlemen stated that if they remained on their home ranches along the river, no such trouble was apparent, but that difficulties were encountered when they moved their cattle into the Hat Creek area.

An old Airedale dog on a Hat Creek ranch was observed to have a large goiter. The condition of this dog is shown in figure 3.

DISCUSSION

The prevalence of simple goiter in certain iodin-deficient areas has been known for decades. Dr. J. W. Kalkus, when he was with the Washington State College, at Pullman, made an extensive investigation of goiter and allied ailments in domesticated animals. The results of his work were published in Bulletin 156, July 1920, under the title, "A Study of Goiter and Associated Conditions of Domestic Animals," in which he stresses the point that if iodin, even in small amounts, is fed or applied to the skin of animals in certain areas deficient in this element, normal births take place.

A comparison of table I, where no iodized salt was given, with table II, where this agent was fed, shows that in the first case 12 out of 16 cows retained their afterbirths, while in the second this condition existed in only one out of 16 animals. In the first instance, out of 16 calves dropped, nine died; in the second, four died of weakness out of 17 dropped. Six of the first lot showed goiter, but none was found in the second herd.

Information gathered at the time of this investigation would indicate that over 100 calves were lost during the period of extremely cold weather, January to March, inclusive. Many were premature, some were hairless and many cows retained the afterbirths. The prematurity of these calves is based on the observation that they did not have any incisor teeth at time of birth—a fulltime calf generally having four—and their small size and extreme weakness.

Observations would indicate that the iodin deficiency is confined principally to Hat Creek, since the trouble does not occur generally on Rising River. This river also has its source in the lava beds, but does not head on Mount Lassen. Due to the fact that cattle grazed in the river area suffer little from this condition, but develop it almost immediately when moved into the Hat Creek area, leads to the suspicion that the Rising River area also may be somewhat deficient and iodized salt should be used there.

Some discussion developed over the amount of iodin necessary to curb this trouble and in what form it should be supplied. The iodized salt that can be purchased on the market contains .023 per cent of potassium iodid, or 2.3 pounds per 1000 pounds of salt. This iodin is incorporated by means of a vacuum process into 5- or 50-pound bricks of pulverized salt. Some stockmen objected to feeding salt in this manner, claiming it made the cows' tongues sore. However, we believe this idea to be exaggerated. One can

realize the difficulties that would be encountered when trying to mix between two and three pounds of potassium iodid in half a ton of salt. It would probably not be properly mixed and the animals would not obtain equal amounts of the element.

We believe that the findings of this investigation emphasize the necessity of having agglutination tests for Bang's disease made before proceeding with control methods relative to handling calving troubles.

During the latter part of March, 1934, another visit was made to this section and it was found that practically all live stock raisers, both on Hat Creek and Rising River, were feeding iodized salt to their cattle. It was reported that several tons had been sold in this area. Very satisfactory results had been obtained, as the former troubles were not encountered this year during the usual bad months of January, February and March.

Crystal Compound Sellers in Trouble

Because they attempted to use to commercial advantage the name of Warm Springs, Ga., by selling what they designated "Warm Springs Crystal Compound," one man has been sentenced to a year in a federal penitentiary and two others are awaiting trial on a charge of conspiring to violate the federal Food and Drugs Act. The "crystals," it was pointed out at the trial, did not come from Warm Springs; nor were they more than a simple laxative, composed of Glauber's salt, similar in action to Epsom salts. The "crystals" cost only a few cents a pound and sold for a dollar a pound. The company and the crystal compound were disavowed in the beginning by the Warm Springs Foundation as having no connection with the Springs, other than that the office was set up in the same town. When the so-called Warm Springs Crystal Compound began to move into interstate commerce, members of the company were seized and indicted by a grand jury.

Specimens Wanted

Mr. Howard R. Hill, Zoölogist, Los Angeles Museum, Exposition Park, Los Angeles, Calif., desires specimens of Linguatulids (pentastomes) for study. These wormlike parasites sometimes are found in the nasal cavities of the dog and fox. Specimens should be placed in 70 per cent alcohol for shipment. Mr. Hill will be pleased to reimburse senders for the postage on all specimens forwarded.

SPONTANEOUS TUBERCULOUS INFECTIONS IN DOGS*

By WILLIAM H. FELDMAN, *Rochester, Minn.*
Division of Experimental Medicine, The Mayo Clinic

The wide distribution of tuberculous infections among domesticated animals is generally recognized. It has been established for many decades that dogs may become victims of tuberculosis, although the malady in these animals is seen so infrequently that opportunity to study the disease in its spontaneous manifestations is seldom encountered. Perhaps the disease occurs more often than published reports would indicate, since it is not unlikely that the salient features of the disease are not recognized, either during the life of the animal or at necropsy.

It has been demonstrated experimentally[1] that the dog possesses a profound resistance to the bacillus of tuberculosis of avian origin, but that it is moderately susceptible to both the human and bovine type of the organism. As one might expect, the provocative agent in the cases reported has been one or the other of these forms of *Mycobacterium tuberculosis*, with a majority of the infections being of human origin and suggesting a significant relationship between the consumptive human being and an intimately associated household pet.

Data on the incidence of spontaneous tuberculosis in dogs are rather meager and pertain largely to information collected by European observers. In France, according to Douville,[2,3] who examined approximately 20,000 dogs, the incidence of tuberculosis is around 4 per cent, although the observations of Petit and Germain, as reported by Lesbouyries,[4] put the incidence as 13.5 per cent. Petit and Germain's figures were based on postmortem examinations of dogs at the Alfort Veterinary School. Information from German sources suggests a somewhat lower incidence. Joest is quoted by Eber[5] as having found an incidence of tuberculosis among dogs at Dresden of 0.83 per cent and Joest is quoted by Rabinowitsch and Kempner[6] as having examined 4,683 dogs in the Berlin pounds, in 1913 and 1914, and having found tuberculosis in 75 (1.6 per cent). None of the affected animals in Joest's series was less than two years of age, although Mihailescu observed the disease in a wolfhound aged five months. In 1920, Joest examined an additional series of 771 dogs and 2.4 per cent of these animals were tuberculous. In Roumania, Mihailescu[7] found, in a period of two years, that 5 per cent of the dogs exam-

*Received for publication, May 2, 1934.

ined at the Bucharest Veterinary College were affected with tuberculosis.

An occasional case has been reported from the United States, and several reports have appeared dealing with the disease as it affects dogs in England. Ramos[8] is credited with the observation that tuberculosis of dogs is quite frequent in the Argentine. In 1,548 necropsies on dogs, reported by Crocker[9] from the Laboratory of Veterinary Pathology of the University of Pennsylvania, tuberculosis was recognized in 15, or approximately 1 per cent.

The paucity of data pertaining to the incidence of canine tuberculosis in the United States makes it unwise to attempt to draw definite conclusions regarding the frequency of the disease. The incidence undoubtedly varies in different localities, and is largely dependent on the existent possibilities for infection from bovine and human sources. Unfortunately many of the published accounts of the occurrence of tuberculosis among dogs have been deficient in that the bacillary type of the infective agent was not determined. An examination of the available literature on the subject has yielded a total of 87 cases in which the type of the organism was determined (table I).

TABLE I—*Cases of spontaneous tuberculosis of dogs in which the bacillary type of Mycobacterium tuberculosis was determined.*

INVESTIGATOR	CASES	TYPE OF ORGANISM		
		BOVINE	HUMAN	ATYPICAL
Schornagel[10]	8	2	4	2
Panisset and Verge[11]	2	..	2	..
Cobbett[12]	17	10	7	..
Smith[13]	1	..	1	..
Rabinowitsch-Kempner[6]	19	1	16	2
Malm[14]	5	5	..	..
Griffith[15]	3	3	..	..
Wilson and Lovell[16]	1	1	..	..
Stableforth[17]	16	6	10	..
Feldman[1,18]	2	1	1	..
Mihailescu[7]	8	..	8	..
Zwick[19]	2	1	1	..
Romer[20]	2	2	..	..
Joest [5,6]	1	1	..	..
Totals	87	33	50	4

In this report I wish to describe two cases of the disease. One of these (case 1) will be summarized briefly, since it was published in detail some years ago.[18] The other (case 2) was observed more recently.

REPORT OF CASES

Case 1 (reported before): An adult bull terrier had progessively lost weight for two months prior to the time he was killed for necropsy. At no time were there symptoms of respiratory embarrassment, although a few days before death the feces had become soft and streaked with blood. Necropsy revealed hemorrhagic colitis and tuberculous involvement of the apical lobe of the left lung. The central portions of the pulmonary lesions had undergone necrosis and, in the resultant cavities, a thin, grayish, sticky, pus-like material was present which revealed acid-fast bacillary forms by direct smears. An emulsion prepared from a portion of the lesion in the lung was used to inoculate guinea pigs, rabbits, and chickens. The results of animal inoculation gave definite evidence of provocative bacteria of the human type.

Microscopically, morbid tissue revealed all transitions from a caseated, somewhat encapsulated, tubercle-like lesion to those that were vigorously active and progressive without the slightest evidence of cellular inhibition. The disease apparently was limited to the lung.

Case 2 (not reported before): An adult male mongrel shepherd of uncertain age had lived under laboratory conditions for two and a half years. The diet had been supervised and had consisted of a mixture of freshly ground horse meat and cornmeal, prepared in a steam cooker, and a commercial dog biscuit. One day a week, a meal of freshly ground raw horse meat had been supplied and, occasionally, condensed pasteurized cow's milk had been fed. Except for occasional periods of exercise, contact with other dogs was impossible, since the animal was confined to a separate cage.

The dog was received from its owner at the laboratory in May, 1931. Information pertaining to the animal's previous history was not available. In October, 1933, being apparently in good health, the dog was selected among others for a study of experimentally produced infection with *Brucella abortus* and, on October 23, being free of specific agglutinins of Brucella, the animal received a raw beef meal to which had been added 1 cc of a suspension of viable *Br. abortus* of swine origin. The raw meat was obtained from a local market and consisted of that grade of meat known as hamburger. Three agglutination tests made during the next five days gave negative results but, on the 16th day after exposure, an agglutination of 1:50 was obtained. Six days later the titre was the same and the dog died on November 20, 26 days after having received the infected meal.

The last few days before death the dog appeared dull and finally exhibited marked respiratory distress.

When the thorax was opened at necropsy, bilateral pleural effusion was noted, the fluid totaling between 300 and 400 cc. Adhesions or other evidence of disease involving either the visceral or parietal pleura were not observed. Near the dorsal surface of the major lobe of the right lung was an indistinctly outlined, indurated area approximately 2 cm in diameter. The lesion was grayish with a slightly yellowish tinge and on cross-section was seen to extend into the parenchyma of the lung to a depth of 1 cm (fig. 1). The lesion was elevated slightly above the surrounding tissue in the lung and contained, near the cortical zone, an irregular cavitation 0.3 to 0.4 cm in diameter. The cavity contained a cloudy, serous-like fluid. The bronchial lymph-nodes seemed

enlarged and charged with black pigment. The left lung was frankly pneumonic, having a solidified consistency. The liver revealed, rather indistinctly, multiple collections of miliary foci of uncertain significance. The only other abnormalities observed were marked thickening of the wall of the urinary bladder and apparent cystitis.

Smears were prepared from the fluid content of the necrotic portion of the lesion in the lung and stained with carbol fuchsin to determine if acid-fast bacteria were present. Many acid-fast bacillary forms were observed, and a portion of the lesion was emulsified for direct culture and for inoculation of animals. Portions of the spleen and one of the mesenteric lymph-nodes also were emulsified in an attempt to demonstrate by culture methods

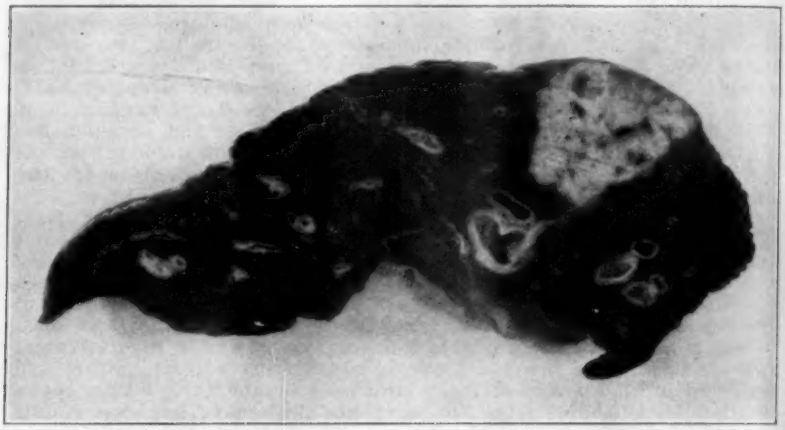

FIG. 1. The tuberculous zone in the lung appears to be proliferative and is elevated slightly above the surrounding pulmonary tissue (case 2).

and animal inoculation the presence of bacteria of the Brucella group, since the dog had been exposed to infection by these organisms some 26 days prior to death.

Direct cultures or animal inoculations failed to disclose *Br. abortus*, although the spleens of all inoculated guinea pigs were emulsified and cultured on a suitable medium. A pure culture of what was considered morphologically to be *M. tuberculosis* was isolated on Herrold's egg-yolk agar seeded with portions of the emulsion prepared from the lesion of the lung (table II).

The tuberculous character of the lesions which developed in the different animals was evident grossly and microscopically and by demonstration of acid-fast bacteria in the diseased tissues in appropriately stained sections and by culture methods. Tubercu-

TABLE II—*Results following inoculation of rabbits and guinea pigs with emulsions prepared from tissues of a dog affected with spontaneous tuberculosis.*

ANIMAL	INOCULUM*	DAYS BEFORE ANIMAL DIED	RESULTS
Rabbit 1	Pulmonary emulsion	44	Aggravated generalized tuberculosis
Rabbit 2	Pulmonary emulsion	10	Negative
Guinea pig 1	Pulmonary emulsion	41	Generalized tuberculosis. Bacilli of tuberculosis cultured from liver
Guinea pig 2	Pulmonary emulsion	26	Generalized tuberculosis. Bacilli of tuberculosis cultured from spleen
Guinea pig 3	Splenic emulsion	26	Purulent diaphragmatic abscess
Guinea pig 4	Splenic emulsion	45	Pleural effusion. Tuberculous spleen and liver
Guinea pig 5	Mesenteric lymph-node emulsion	44	Pneumonia. No tuberculosis
Guinea pig 6	Mesenteric lymph-node emulsion	10	Negative

*Rabbits inoculated intravenously, guinea pigs subcutaneously.

losis developed in animals receiving pulmonary and splenic emulsions, respectively; in animals which received the inoculum prepared from the mesenteric lymph-node, however, the disease failed to develop.

The infective agent exhibited a virulent, malignant type of pathogenicity for both rabbits and guinea pigs and grew on artificial media only sparingly. The organism was therefore considered to be of the bovine type.

HISTOPATHOLOGY OF THE LESIONS

All tissues were fixed in a neutral 10 per cent solution of formalin, embedded in paraffin, and sections obtained in duplicate; one set was stained with hematoxylin and eosin and the other with carbol fuchsin.

Left lung: Diffuse exudative pneumonitis was present with profuse and richly cellular bronchial exudation that occluded most of the larger respiratory passages. A marked, subserous, cellular infiltration, which extended to the surface of the visceral pleura in a few instances, suggested pleuritis of some duration. Deposits of particles of carbon were observed in the vicinity of the smaller bronchioles. Two small cysts containing the unidentified remains of an animal parasite were seen in the midst of one

of the pneumonic zones. No evidence of tuberculous infection was disclosed.

Right lung: The tuberculous zone consisted essentially of a conglomeration of mononuclear cells and a few polymorphonuclear leucocytes and lymphocytes arranged in indistinct alveolar-like packets. The morbid process extended to the surface of the lung and, at one place, the continuity of the superficial covering of the lung had been broken by underlying necrobiosis which was an impressive part of the specific lesion. Necrosis was largely coagulative, and extended extensively throughout most of the lesion. Architectural patterns, suggestive of so-called tubercles, were not apparent, nor were giant-cells of the Langhans type (fig. 2). Encapsulation of the lesion had not occurred. In fact, there was not the slightest inhibitory handicap of an anatomic character to be seen anywhere, the process expanding peripherally in an ever-widening fashion. The adjacent bronchioles and bronchi were occluded by mucus-like material. Mineral salts were not seen. The balance of the parenchyma of the lung revealed a more or less diffuse type of pneumonitis which was probably terminal. Acid-fast bacillary forms were readily demonstrated in the necrotic débris throughout most of the lesions. One large bronchus was filled with multiple cystic structures containing what were considered to be remains of nematodes. These structures were so numerous and the resultant tissue reaction so vigorous that this particular respiratory passage was entirely occluded.

Spleen: There was no significant histologic alteration of the spleen, although, curiously enough, a careful search disclosed not a few acid-fast bacillary forms, often arranged in clumps but appearing occasionally alone.

Liver: The multiple miliary foci observed in the liver macroscopically proved histologically to be composed largely of mononuclear cells or histiocytes and a few lymphocytes. The cytoplasm of many of the cells appeared clear or foamy (fig. 3). The peripheral reaction was minimal in amount and central necrosis had not become evident. Sections of the liver were stained by Pap's method to reveal the reticular structure of the cells of respective focal lesions. These argentophilic fibrils, which are characteristically present in the typical, well-formed tubercle, seldom were seen in lesions of the liver, and their absence suggests immaturity of these focal cellular reactions.

Kidneys: Both kidneys gave evidence of chronic glomerular changes of a cirrhotic character, but specific lesions of tuberculosis did not occur. In the lateral aspect of the cortex of one

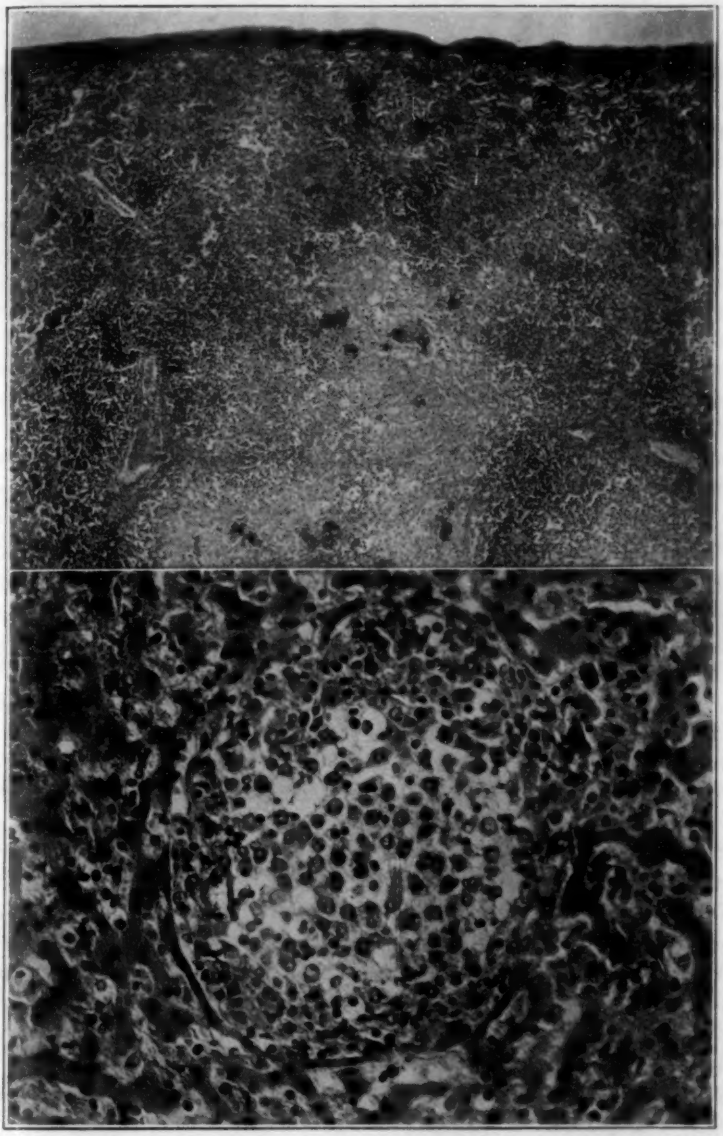

FIG. 2 (above). Section through the tuberculous nodule of the lung (case 2.) The reaction is of a diffuse character with extensive necrosis and no encapsulation. Acid-fast bacteria were present in the cellular débris (x 45).

FIG. 3 (below). Solitary tubercle of the liver (case 2). Many of the epithelioid cells have a clear or foamy appearance (x 250).

kidney, functional derangement was rather extensive and profound. The fused, rather acellular, hyaline appearance of many of the glomeruli in this portion of the organ suggested amyloid degeneration, but this substance could not be demonstrated by specific stains. Many of the tubules contained casts, and pyelonephritis was evident in each organ.

Urinary bladder: Although there was marked hyperplasia of the muscular portions of the wall, the mucosa appeared normal.

To summarize the pathologic changes the following may be listed: The lungs revealed a solitary progressive type of tuberculous lesion, associated with exudative pneumonitis and parasitism of one of the larger bronchi. The liver contained multiple, miliary, cellular foci without necrosis or encapsulation but containing acid-fast bacteria. Histologically, the spleen was normal. The kidneys revealed a chronic, nonspecific, nephritic change largely glomerular in character. In no instance were the tuberculous lesions calcified.*

COMMENT

The histopathology of the tuberculous lesions in this case is not that of a chronic process of prolonged duration. Instead, the converse is apparently true and, after a consideration of the character of the respective tissue reactions, one cannot avoid the impression that the process was progressively acute and the duration of the disease perhaps a few or several weeks rather than months or years. The lesions in the liver and the demonstration of bacilli of tuberculosis in the spleen probably represent a recent hematogenous dissemination of infective bacilli from the primary lesion in the lung. The lesions of the liver are definitely those of short duration and resemble tissue reactions in the liver following intravenous injection of bacilli of tuberculosis experimentally. The lesion in the lung must be considered of recent inception for the following reasons: (1) predominance of mononuclear cells or histiocytes with few if any epitheliod forms, (2) entire absence of connective tissue encapsulation, (3) character of necrobiosis which was of a coagulative nature in contrast to the caseous character of the usual lesions of chronic tuberculosis, and (4) aggressive behavior of the pulmonary lesion, as revealed by its diffuse local expansion and by the recent dissemination of the infection to the abdominal viscera.

If the assumption that the disease was of recent origin be correct, the question as to the source of infection becomes signifi-

*Nieberle and Pallaske[21] consider the absence of calcification one of the characteristics of tuberculous lesions in dogs.

cant. Since the affected animal had been maintained under laboratory conditions for approximately two and a half years before it was used for the experiment pertaining to infection with Brucella, the possibilities for contact with the organism of tuberculosis during this time should be examined. It seems entirely unlikely that the infection could have occurred as a consequence of ingestion of the commercial dog bread or the cooked horse meat-corn meal mash. The meal of uncooked horse meat fed once weekly may have contained the infective bacteria, since horses rarely are affected with tuberculosis and in practically all instances the type of infective organism has been bovine. Tuberculosis among horses, however, usually is characterized by gross lesions of striking appearance, and such lesions would have been recognized when the animal in this case was examined at necropsy. In spite of the large number of horses slaughtered and subsequently consumed cooked or uncooked by the dogs in our laboratory, no instance of equine tuberculosis has yet been encountered. Furthermore, the fact that only the skeletal bones and the musculature are used would reduce the possibility of infection even though the horse had had tuberculosis. Another possible source of tuberculous infection in this dog was the ground beef or hamburger. The dog was fasted for 24 hours prior to being fed the meat meal, which fact should favor the invasion of tissues by organisms that may have been present. Tuberculosis of cattle is not uncommon and the possibility that the meat in question may have been obtained from a concern whose products were not subjected to veterinary inspection are factors worthy of consideration. Lastly, the age of the lesion of the lung would coincide in a general way with the time which elapsed between the date of the meal of hamburger and the death of the animal (26 days).

Although none of the explanations as to the source of the infection are capable of proof, the last-mentioned one seems reasonable and the most likely. However, one must admit of the possible existence of other sources of the disease the exact character of which is uncertain and obscure, and for this reason it would be unwise and perhaps fallacious to fix definitely the source of infection.

The experience of many observers has established certain facts concerning tuberculosis in dogs as the disease occurs spontaneously. The clinical evidence seems to substantiate the experimental observations of Leudet and Petit[22] that dogs are rather refractory to tuberculous infection by way of the respiratory tract and that infection of the viscera occurs as a consequence

of the organism entering the body through the intestines. Of the reported cases of spontaneous tuberculosis in dogs in which the bacillary type of infection has been found, in the greatest number the disease has been due to bacilli of human origin. This indicates that the possibility of infection with this form of the organism is greater than with the bovine type. Dogs not infrequently are associated in the same household with consumptive human beings, and the tendency for dogs to ingest expectorated sputum that has been improperly disposed of is well known. The probable reason why the type of organism that occurs in man is responsible for more cases of canine tuberculosis in certain localities than the bovine type undoubtedly is because of this greater opportunity that exists for exposure of the animals to the bacilli of tuberculosis of man. Experimentally, there is no evidence that dogs are more susceptible to the human form of the infective agent than to those of bovine origin. The avian variety of the bacillus is not concerned in the pathogenesis of tuberculosis in dogs. Experimental observations[1] have shown the dog to be extremely resistant to tuberculous infection of chickens, and spontaneous tuberculosis of the dog due to the avian type of the organism has not to my knowledge been reported.

The distribution of the lesions in spontaneous tuberculosis of dogs is of some interest. In 22 cases reported by various observers, the lesions were distributed anatomically as follows: lungs, 18 cases; liver, 11 cases; kidneys, 9 cases, mediastinum and pericardium, 6 cases; pleura, 5 cases; mesenteric lymph-nodes, 3 cases; pancreas, 2 cases, and peritoneum, 2 cases. These observations, although relatively few in number, indicate the predilection of the lung of the dog for the infective agent. In the great majority of cases this organ is the situation of the primary focus. Most of the other tissues and organs would seem to become infected secondarily as a consequence of bacteriemia, with the liver and kidneys usually developing multiple lesions of a miliary character.

In the recognition of canine tuberculosis in the living animal there are few symptoms that are characteristic. The affected animals suffer marked loss of weight, progressive weakness, and respiratory distress, with occasional paroxysmal coughing. Pleural effusion is not uncommon and, when present, adds to the animal's distress. The presence or absence of fever is not a reliable criterion and, unfortunately, the tuberculin test is not a dependable procedure in the diagnosis of the disease in dogs.

Dogs affected with tuberculosis must be looked on as a distinct hazard to the health of man. The predilection of the infective

organism for the lungs and the character of the resultant lesions provide a circumstance that enhances the possibility for dissemination of the infective material. The lesions commonly undergo necrosis, and the extension of the bacteria-laden cellular detritus into the larger respiratory passages makes the pulmonary exudate from such an animal a potential menace to human beings, particularly to children who frequently fondle dogs in a very intimate way.

SUMMARY

A case of spontaneous tuberculosis in a dog kept under laboratory conditions is described. The infection was due to the bovine type of the organism. The primary lesion was in the lung with secondary involvement of the liver.

REFERENCES

[1]Feldman, W. H.: The pathogenicity for dogs of bacilli of avian tuberculosis. Jour. A. V. M. A., lxxvi (1930), n. s. 29 (3), pp. 399-419.
[2]Douville: Cited by Calmette, A.: Tubercle Bacillus Infection and Tuberculosis in Man and Animals. (Williams and Wilkins, Baltimore, 1923), p. 358.
[3]Douville: Cited by Eber.
[4]Lesbouyries, S.: La Tuberculose des Carnivores Domestiques. (Vigot Frères, Paris, France, 1926.)
[5]Eber, A.: Die Tuberkulose der Tiere. Bericht über die Jahre 1905-1914. Ergeb. d. all. Path. u. path. Anat., xviii (1917), pp. 1-371.
[6]Rabinowitsch-Kempner, L.: Domestic animals as sources of tuberculous infection. Abst. in Amer. Rev. Tuber., vi (1922), pp. 151-152.
[7]Mihailescu, M.: Contribution à l'étude de la tuberculose canine en Roumanie. Rev. Gén. de Méd. Vét., xxxviii (1929), pp. 65-77.
[8]Ramos: Cited by Eber.
[9]Crocker, W. J.: Three thousand autopsies. Corn. Vet., ix (1919), pp. 140-161.
[10]Schornagel: Cited by Panisset and Verge.
[11]Panisset, L., and Verge, J.: Origine humaine de Bacilli de Koch dans deux cas de tuberculose canine. Compt. Rend. Soc. Biol., xc (1924), pp. 341-342.
[12]Cobbett, L.: The rôle of the three types of tubercle bacilli in human and animal tuberculosis. Lancet, i (1922), pp. 979-983.
[13]Smith, T.: Studies in mammalian tubercle bacilli. III. Description of a bovine bacillus from the human body, a culture test for distinguishing the human from the bovine type of bacilli. Jour. Med. Res., xiii (1905), pp. 253-300.
[14]Malm: Cited by Griffith.
[15]Griffith, A. S.: Tuberculosis of the domesticated species of animals. Jour. Comp. Path. & Therap., xli (1928), pp. 53-75; 109-122.
[16]Wilson, G. S., and Lovell, R.: Tuberculosis in a dog due to infection with a bovine virus. Jour. Path. & Bact., xxxi (1928), pp. 583-584.
[17]Stableforth, A. W.: A bacteriological investigation of cases of tuberculosis in five cats, sixteen dogs, a parrot, and a wallaby. Jour. Comp. Path. & Therap., xlii (1929), pp. 163-188.
[18]Feldman, W. H.: Spontaneous tuberculosis in a dog. Proc. Staff Meetings Mayo Clinic, v (1930), pp. 78-80.
[19]Zwick: Quoted by Eber.
[20]Römer: Quoted by Eber.
[21]Nieberle, K., and Pallaske, G.: Studien zur pathologischen Anatomie und Pathogenese der Tuberkulose der Haustiere. VI. Mitteilung. Die Tuberkulose der Fleischfresser. Arch. f. Wiss. prakt. Tier., lxiv (1931), pp. 181-207.
[22]Leudet and Petit, G.: Résultats d'expériences d'inoculation de la tuberculose humaine au chien, infection naturelle de ce dernier par les voies digestives. Rec. de Méd. Vét., lxxxi (1904), pp. 301-307.

Prosperity is not without many fears and distastes; and adversity is not without comforts and hopes.—FRANCIS BACON.

RESULTS OF COMPULSORY TESTS FOR BANG'S DISEASE*

By B. J. KILLHAM, *East Lansing, Mich.*
Extension Specialist in Animal Pathology
Michigan State College

An ordinance requiring that all milk sold in the city of Jackson, Mich., must come from cows which have passed an approved blood-test for Bang's disease was made effective during the fall of 1933. The testing was begun immediately and completed as rapidly as conditions permitted. The blood samples were collected by five practicing veterinarians, and the tests were made in the laboratory of the Department of Animal Pathology, Michigan State College.

Complete herd tests were not required, but in a number of instances all of the cattle over six months of age were included. The ordinance is so constructed as to require the test of all female cattle over one year of age which are included in the herds of Jackson City producers. The herds tested were larger than the average for Jackson County and, from the standpoints of production and sanitary surroundings, better than the average.

All of the cattle tested in the project were located in Jackson County. The distribution over the county was fairly uniform, and 18 of the 19 townships in the county were represented. The lone exception was a township not heavily populated with cattle, located in the extreme corner of the county.

The first complete tuberculin test of the cattle of Jackson County, in 1922, revealed 24,937 animals. This included cattle of all breeds, types and ages. It is estimated that the total cattle population of the county now is 26,500. The federal census for 1930 gives the number of cows and heifers in Jackson County, kept mainly for milk-production, as 13,145. Nearly all of the 4,382 cattle tested because of the compulsory ordinance were included in this group, which numbered 13,145 in 1930, and, hence, represent approximately one-third of the cows and heifers kept for milk-production.

Previous attempts to estimate the prevalence of Bang's disease, with a few notable exceptions, have been based upon results obtained from blood samples submitted for test. In most instances the samples were sent in for test because disease was suspected, herds were in the process of accreditation, sales were

*Received for publication, July 6, 1934.

to be consummated, cattle were to be shipped interstate, or the test was desired for some other reason of interest to the cattle-owner. The Jackson County situation presents a compulsory test which included approximately one-third of the cows and heifers kept for milk-production, and so distributed as to represent a very good cross-section of the county with respect to Bang's disease. The percentage of infection among the tested cattle would undoubtedly be decreased through including more young cattle in the tests, but it is not at all probable that the alarmingly high percentage of herd infection would be materially affected by the inclusion of the younger animals.

In theorizing upon the possible relation of the situation in Jackson County to Michigan as a whole, some of the data pertaining to tuberculosis have been reviewed. The first complete tuberculin test of the cattle of Jackson County showed 5.25 per cent infection. The first complete test of the cattle in the Lower Peninsula revealed approximately 4.0 per cent of the cattle to be tuberculous. Presumably the opportunities for the development and spread of Bang's disease in Jackson County have been approximately equivalent to those presented for tuberculosis. If so, it might be reasonable to expect the cattle of the Lower Peninsula as a whole to be infected with Bang's disease to the extent of about 75 per cent of the incidence shown for Jackson County.

Table I shows the results of agglutination tests applied to Jackson County cattle as compared with the results reported by Fitch and Donham[1] for two Minnesota townships.

TABLE I—*Results of individual tests for Bang's disease in Minnesota and Michigan.*

DIAGNOSIS	CATTLE TESTED			
	TWO MINNESOTA TOWNSHIPS		JACKSON COUNTY, MICHIGAN	
	No.	PER CENT	No.	PER CENT
Negative	2,811	88.3	3,621	82.6
Suspect	188	5.9	292	6.7
Positive	184	5.8	469	10.7
Totals	3,183	100.0	4,382	100.0

Much of the difference in the results obtained in the two states could probably be attributed to the fact that in Jackson County, Mich., in many instances the younger cattle, which usually show a lighter infection, were not included in the test.

Table II continues the comparison through indicating the results from the standpoint of herds tested.

TABLE II—*Results of herd tests for Bang's disease in Minnesota and Michigan.*

DIAGNOSIS	HERDS TESTED			
	TWO MINNESOTA TOWNSHIPS		JACKSON COUNTY, MICHIGAN	
	No.	PER CENT	No.	PER CENT
Negative	120	66.3	112	38.1
Suspect*	23	12.7	52	17.7
Positive	38	21.0	130	44.2
Totals	181	100.0	294	100.0

*Herds with 1 or more suspects; none positive.

The difference now becomes more apparent. The percentage of infected herds in Jackson County is more than twice that exhibited for the two Minnesota townships. Even though herds which show suspects only are disregarded, and it is probable that most of these herds are not infected, there still remains the fact that nearly one-half of the herds contained one or more infected animals.

Undoubtedly the observation recorded by Birch,[2] that "the survey not only confirms the universal observation that it is the large herd, and the one that depends on outside sources for replacements, that suffers most from Bang's disease, but it reveals something of the degree in which these influences operate," is pertinent to Jackson County. Although the average number of animals for each herd tested was about 15, not all of the young cattle were included, and, hence, the herds would average well over 20 animals and thus qualify as large herds in Michigan. The production of whole milk for city distribution also presents the need for keeping the quantity of milk up to a prescribed amount, and this need at least presents the temptation to acquire additional cattle from outside sources.

TABLE III—*Distribution of reactors in 130 herds.*

	REACTORS IN HERD					
	1	2	3	4	5	6 (or more)
Herds	44	24	14	9	13	26

The prospect is not alarming when viewed from the aspect of the extent of the disease in the infected herds. Table III indicates briefly the distribution of the reacting animals in the 130 infected herds.

Considered from this angle, the outlook is far from discouraging. The difficulty of eradicating Bang's disease from a given herd will depend upon a number of factors, but probably it can be asserted safely that in general the herds containing one, two, or three reactors do not present a hard problem and usually are cleaned up easily. Under some conditions herds containing four or five reactors might be included in the same group. But, if only those herds containing not more than three reactors are considered, the removal of the 82 herds in that class from the group of infected herds, which comprised 44 per cent of the herds tested, would reduce to 16 per cent of the whole the herds in which there are serious control or eradication problems. In this particular instance practically all owners of infected herds have been visited and advised as to control measures, and it is believed that the restrictions which were placed with a view to protecting human health have initiated a work which will have a very wholesome effect insofar as disease control among cattle is concerned.

The visits to the owners of the infected herds in Jackson County have served to emphasize two facts—one new and the other old. Despite the fact that this was the first effort in Michigan to require the blood-test for all cows supplying milk to a given city, practically no evidence of antagonism was observed among the owners of the diseased cattle, and many of these farmers had disposed of their reactors or were preparing to do so without any prospect of indemnity. Judging from the contact that was had with some owners of clean herds, it was not considered at all probable that any complaint would be received from that source.

The other fact was that the act of abortion is being too strongly emphasized with reference to Bang's disease. That the disease causes cows to abort is appreciated; but there are affected cows which do not abort, others which apparently do not abort for some time after becoming infected, and many others which abort once or possibly twice and then produce calves.

Accurate data regarding abortions are rarely available on the average farm, but the careful, conscientious farmer has a fair idea of his situation in this respect. A number of infected herds were encountered, some infected to the extent of 50 or 75 per

cent, in which, if the statements of the owners, most of whom appeared to be honest, can be accepted, there had been practically no abortions for periods varying from two to five years. Of course, with herds tested for the first time, it was not possible to determine definitely how long the infection had been present, except in a few instances, but it seems reasonable to assume that many of the herds had been infected for several years unbeknown to the owners because they were using a false gauge—the abortion.

ACKNOWLEDGMENTS

The author gratefully acknowledges the help in collecting data which was given by Dr. E. J. MacLachlan, of the Jackson Department of Health, and S. J. Culver, Agricultural Agent for Jackson County.

REFERENCES

[1]Fitch, C. P., and Donham, C. R.: Survey to determine the incidence of Bang's disease in Minnesota cattle. Proc. Soc. Exp. Biol. & Med., xxx (1933), 9, p. 1203.

[2]Birch, R. R.: A Bang's disease survey of a representative dairy township. Jour. A. V. M. A., lxxxiv (1934), n. s. 37 (6), pp. 854-862.

Civil Service Examination

The U. S. Civil Service Commission announces an open competitive examination for "Junior Parasitologist." Applications for the position must be on file with the Commission not later than November 26, 1934.

At present, there is a vacancy in the U. S. Bureau of Animal Industry, with headquarters at Beltsville, Md. The Department of Agriculture wishes to fill this position with a woman eligible. Both men and women, who are qualified, will be admitted to the examination. The entrance salary is $2,000 a year, subject to a deduction of not to exceed 5 per cent during the fiscal year ending June 30, 1935, as a measure of economy, and also to a deduction of 3½ per cent toward a retirement annuity. The duties are to conduct research in the field of animal parasitology, including such routine assignments as the identification of parasites, the examination of animals for parasites, and similar work.

Full information may be obtained from the Secretary of the U. S. Civil Service Board of Examiners at the post office or customhouse in any city which has a post office of the first or the second class, or from the U. S. Civil Service Commission, Washington, D. C.

Extreme remedies are very appropriate for extreme diseases.—HIPPOCRATES.

PRELIMINARY STUDIES ON THE COMPLEMENT-FIXATION TEST FOR THE DIAGNOSIS OF BOVINE ANAPLASMOSIS*

By CHARLES W. REES, *Zoölogical Division*
and WILLIAM M. MOHLER, *Pathological Division*
U. S. Bureau of Animal Industry, Washington, D. C.

During the last several years, the Pathological and Zoölogical divisions of the Bureau of Animal Industry have been engaged in a study of the various phases of anaplasmosis. In recent years, attention has been paid to the possible use of the complement-fixation test as a means of detecting the disease in its acute and carrier stages.

Numerous attempts in the Pathological Division to prepare antigen for complement-fixation tests from spleens, livers and other organs of infected animals were unsuccessful. However, definite antigenic value was demonstrated in the washed centrifugate of distilled-water-laked blood drawn from a splenectomized cow during the height of the anaplasmosis reaction.[1] While the results obtained were encouraging, sufficient quantities of this type of antigen could not be produced for extensive study.

With the knowledge that complement fixation was possible, and since there was available a supply of infected ticks in various stages, attention was directed to the possibility of infected ticks serving as a source of antigen.

It has been demonstrated that anaplasmosis can be transmitted experimentally by a number of ticks, including *Rhipicephalus sanguineus*,[2] *Dermacentor variabilis*,[3, 4] *D. andersoni*,[5] and *Boöphilus annulatus*.[5, 6] Results obtained thus far with antigens prepared from infected ticks *(R. sanguineus and B. annulatus)* appear to warrant this preliminary report.

METHODS OF PROCEDURE

One species of three-host tick, *R. sanguineus*, was used. These ticks were confined in cloth bags which were fastened by adhesive plaster to the scrotums of bulls or the ears of rabbits. These bags were required to collect the larvae, nymphs and adults, respectively, which otherwise would drop to the ground after engorgement, and be lost in the stable litter. With the one-host tick, *B. annulatus*, the bags were not required. Titrations were made of antigens of larvae, nymphs and adults of *R. sanguineus*, both engorged and unengorged. The engorged ticks were used

*Received for publication, September 24, 1934.

as test material either immediately or at stated intervals after engorgement. Tests were made of antigens prepared from engorged and unengorged nymphs that had engorged as larvae either (1) on rabbits, (2) on susceptible cattle, or (3) on cattle having clinical anaplasmosis; also on adults that engorged as nymphs under the above-mentioned conditions. The tested *B. annulatus* antigens were of ova, unengorged larvae, and engorged females, these ticks having been collected from cattle in Florida and Louisiana.

TITRATION OF THE TICK ANTIGENS

From February 28 to March 5, 1934, nymphs of *R. sanguineus* engorged on a splenectomized bull (109) that had clinical anaplasmosis. On March 14, by the method of Cowdry and Ham[7] the exoskeletons of 16 of the nymphs were removed, and the viscera and exoskeletons were triturated separately, and each mass was taken up in 12 cc of normal saline, furnishing antigens 1 and 2, respectively. Titrations were made of these antigens against the serum of heifer (116), a clinical case of anaplasmosis, and the serum of a normal bovine (119). Antigen 1 gave definite evidence of antigenic properties, whereas antigen 2 showed no antigenic value whatever.

On March 15, a normal saline extract (25 cc), antigen 3, was made from 25 nymphs (viscera only), and a distilled-water extract (25 cc), antigen 4, was made from 25 other nymphs. On March 15, both antigens were tested; the results are given in tables Ia and Ib.

TABLE Ia—*Titration of antigen 3, a saline extract of R. sanguineus (infected nymphs), against anaplasmosis serum.*

SERUM	ANTIGEN 3								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	4+	4+	4+	4+	4+	4+	4+	4+	−
Negative (119).....	−	2+	3+	4+	4+	4+	4+	4+	−

Key: 4 + = 100 per cent inhibition of hemolysis; strongly positive.
3 + = 75 per cent inhibition of hemolysis; moderately positive.
2 + = 50 per cent inhibition of hemolysis; weakly positive.
1 + = 25 per cent inhibition of hemolysis; very weakly positive.
— = Complete hemolysis, or negative.

On March 28, the remaining nymphs had molted to adults and some of them were permitted to engorge on the susceptible bull (119). On April 5, twelve males and six partly engorged females were recovered, and the specimens of each sex were dissected

TABLE Ib—*Titration of antigen 4, a distilled water extract of R. sanguineus (infected nymphs), against anaplasmosis serum.*

SERUM	ANTIGEN 4								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	1+	2+	3+	4+	4+	4+	4+	4+	−
Negative (119)....	−	−	−	−	−	−	−	−	−

separately in 12 cc of distilled water. These antigens, 5 and 6 respectively, were kept in the refrigerator and occasionally shaken in a machine until April 11.* Results obtained with antigens 5 and 6 are given in tables IIa and IIb.

TABLE IIa—*Titration of antigen 5, a distilled water extract of infected male R. sanguineus, after engorging on normal bull, against anaplasmosis serum.*

SERUM	ANTIGEN 5								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	−	1+	2+	3+	4+	4+	4+	4+	−
Negative (119).....	−	−	−	−	−	−	−	−	−

TABLE IIb—*Titration of antigen 6, distilled water extract of infected female R. sanguineus, after engorging on normal bull, against anaplasmosis serum.*

SERUM	ANTIGEN 6								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	−	1+	2+	3+	4+	4+	4+	4+	−
Negative (119).....	−	−	−	−	−	−	−	4+	−

On April 6, other nymphs that engorged on bull 109, an infected animal, were placed on a rabbit. On April 20, 19 males and nine partly engorged females taken off this rabbit were dissected and prepared as before in distilled water for use as antigens 7 and 8, respectively. Fairly good fixation was obtained with these extracts, but the antigenic unit was higher than in the previous tests. Results are given in tables IIIa and IIIb.

On April 6 to 13, ticks that had engorged as larvae and as nymphs on rabbits were permitted to engorge as adults on rabbits.

*On May 4, bull 119 reacted to anaplasmosis, thus demonstrating that the active etiologic agent was present in the ticks at the time of engorgement.

TABLE IIIa—*Titration of antigen 7, distilled water extract of infected male R. sanguineus, after engorging on rabbit, against anaplasmosis serum.*

SERUM	ANTIGEN 7								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	−	−	−	2+	2+	4+	4+	4+	−
Negative (119).....	−	−	−	−	−	−	−	−	−

TABLE IIIb—*Titration of antigen 8, distilled water extract of infected female R. sanguineus, after engorging on rabbit, against anaplasmosis serum.*

SERUM	ANTIGEN 8								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	−	−	−	1+	3+	4+	4+	4+	−
Negative (119).....	−	−	−	−	−	−	−	−	−

On April 13, test antigens were prepared as before with these ticks. The complement-fixation test was negative. It appears, therefore, that no fixation occurred unless the ticks were infected with the etiological agent.

A number of antigens were prepared from the specimens of *B. annulatus* that were sent by the field workers* from Florida and Louisiana. By the use of the serum of heifer 116, a case of pure anaplasmosis and also of the serum of a recovered field case of piroplasmosis,† complement fixation was demonstrated with these antigens.

On May 2, antigen 9 was prepared from 16 engorged female cattle ticks by triturating them in 26 cc of distilled water. A dilution of this test antigen with distilled water, 1 part to 12 parts, was made and tested with the results shown in table IV.

Against the anaplasmosis serum of heifer 116, perfect fixation was obtained with 0.4 cc of antigen, whereas 1.0 cc of antigen against negative serum showed no anticomplementary action.

Against the piroplasmosis-positive serum from the field case mentioned above, perfect fixation was obtained with 0.05 cc of this antigen, whereas 1 cc of the antigen against normal serum showed no anticomplementary action, as shown in table V.

*Forwarded by Dr. T. W. Cole, Jacksonville, Fla.

†It cannot be stated definitely at this time that this animal may not have had a previous anaplasmosis infection.

TABLE IV—*Titration of antigen 9, distilled water extract of B. annulatus, infected engorged females, against anaplasmosis serum.*

SERUM	ANTIGEN 9								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (116).....	−	−	2+	3+	4+	4+	4+	4+	−
Negative (119).....	−	−	−	−	−	−	−	−	−

On May 4 a number of *B. annulatus* larvae, the progeny of female ticks sent in from the field, were placed on bull 119. As has been noted, this bull had reacted to anaplasmosis. At this time he was convalescing, the temperature having subsided to

TABLE V—*Titration of antigen 9, distilled water extract of B. annulatus, infected engorged females, against piroplasmosis serum.*

SERUM	ANTIGEN 9								CONTROL
	.05	0.1	0.2	0.3	0.4	0.5	0.8	1.0	
Positive (field case).	4+	4+	4+	4+	4+	4+	4+	4+	−
Negative (119).....	−	−	−	−	−	−	−	−	−

normal. On May 10, the bull had a temperature of 103° F. and the fever persisted for several days, but no parasites were demonstrated in the smears. On May 16, about 35 cc of citrated blood from this bull was injected into bull 121. This bull reacted with a temperature of 104° F. on May 25, and *Babesia bigemina* was demonstrated in the smears. Therefore, this parasite was present in these seed ticks. Bull 121 reacted to anaplasmosis on May 31. Forty of these same larvae were made into an antigen with 10 cc of distilled water. This antigen gave definite fixation with the serum of heifer 116 that had clinical anaplasmosis at the time the serum was drawn but was known to be free from piroplasmosis. However, we can not definitely rule out the presence of anaplasma in the ticks. The same type of reaction was obtained with this antigen when tested against the serum of the field case of piroplasmosis.

CONCLUSIONS

The following statements appear to be supported by the experimental data of the present paper:

1. Antigen prepared from nymphs and adults of *Rhipicephalus sanguineus* known to harbor the etiological agent gave positive complement fixation against a serum from an active case of anaplasmosis.

2. The antigen was demonstrable in nymphs that engorged on a clinical case, and the antigenic properties appeared to increase during the engorgement either on susceptible bovines or on rabbits of adults that molted from these nymphs.

3. The antigen did not appear to be present in adults that engorged as larvae, nymphs and adults on rabbits.

4. Antigenic value was demonstrable by complement fixation with engorged females of *Boöphilus annulatus* obtained from tick-fever-affected cattle against both pure anaplasmosis serum and against serum of an animal known to be affected with piroplasmosis but from which anaplasmosis could not be excluded absolutely. Infected larvae of this species showed similar antigenic value.

5. These studies indicate the possibility of developing a serological diagnostic method for detecting carriers of the disease.

REFERENCES

[1]Mohler, J. R.: Report of the Chief of the U. S. Bureau of Animal Industry, 1931-32, p. 43.
[2]Rees, C. W.: The experimental transmission of anaplasmosis by *Rhipicephalus sanguineus*. No. Amer. Vet., xi (1930), 9, pp. 17-20.
[3]The experimental transmission of anaplasmosis by *Dermacentor variabilis*. Sci., n. s. lxxv (1932), pp. 318-320.
[4]Rees, C. W.: The experimental transmission of anaplasmosis by *Dermacentor andersoni*. Parasitol., xxiv (1932), pp. 569-573.
[5]Transmission of anaplasmosis by various species of ticks. U. S. Dept. of Agr. Bul. 418 (1934), pp. 1-17. Illus.
[6]Dikmans, G.: Anaplasmosis. IV. The carrier problem. Jour. A. V. M. A., lxxxii (1933), n. s. 35 (6), pp. 862-870.
[7]Cowdry, E. V., and Ham, A. W.: Studies on East Coast fever. I. The life cycle of the parasite in ticks. Parasitol., xxiv (1932), 1, pp. 1-49.

Liver Fluke Control

Since the prolonged drouth has dried up the breeding places of the small water snail that is responsible for the early development of the liver fluke, the chances for eradicating this pest from the farm altogether are excellent, according to the *Journal of the Ministry of Agriculture*, of England. The few remaining haunts of the snail, which are centers of infection for the liver fluke, should be dressed with a mixture of finely powdered bluestone and dry sand, in the proportions of 1 to 4, and at the rate of 1¼ to 2¼ cwt. to the acre. The treatment may be repeated after an interval of three weeks. It is wise to keep sheep away from the treated ground until after rain has fallen. As a further step toward eradicating the parasite, the treatment of all adult sheep with carbon tetrachlorid or, alternatively, with extract of male fern, is recommended.

Veterinary Record.

THE SURGICAL REPAIR OF A DIAPHRAGMATIC HERNIA IN A DOG*

By RUDOLPH H. SCHNEIDER, *Boston, Mass.*
Angell Memorial Animal Hospital

The purpose of this article is not merely to render an account of a rare, if not singular success, relative to a dog which is fully restored to its normal state of health and activity by reason of the surgical repair of a ruptured diaphragm, but to stress such essence as the practical aspect of the intervention, the possibilities of even the most modestly equipped surgery and the significance of earnest endeavor.

The subject of discourse is "George," a 5-year-old Sealyham terrier, summering with his mistress, Mrs. G., at Vineyard Haven, Mass. George was presented for examination several days after having been stricken with what was described as a sudden attack of labored breathing, which was aggravated to the point of asphyxia by even moderate exertion. The cause for the delayed visitation to the hospital was not disclosed, although the symptoms were clearly alarming enough to demand immediate counsel. To be sure, the gravity of the situation was unsuspected.

However, a glancing inspection, together with the knowledge of the sudden onset of events, immediately inspired the suspicion of a diaphragmatic hernia, obviously, of traumatic origin. The characteristically short, stifled respiration, blended with the large area of dull percussion over the chest; the subdued vesicular murmurs and the gauntness of the abdomen connoted a diagnosis of rupture, with the prolapse of abdominal viscera into the thorax. These facts were substantiated by subsequent radiographic examination, following a bismuth meal, which revealed that the stomach remained in approximate situ, that the liver shadow was thrust forward, that intestinal loops were intruding into the chest as far forward as the sixth rib, and that it was reasonably certain to be of a right-sided nature.

*Received for publication, September 13, 1934.

Upon this confirmation the fate of George was still to be decided. To be sure, he could not have lived a normal life as he was, nor was it the wish of his mistress that he should, yet, she was reluctant in consigning him to a lethal fate without a fair trial. This attitude was, indeed, gratifying to the writer, who had long sought the occasion and the justification for making an inroad into this uncanny surgical field. Thereupon, a rather bold prognostication was made, to force the issue, but of liberal and seemingly sufficient latitude to impress the owner of the significance of the proposed intervention. The subsequent success of this moral obligation not only meant the sparing of a prized possession but bears out the maxim, "Something attempted, something done," and the fact that one should not rely wholly upon tradition and the assumption of impossibilities. This is a new era of "new dealing" and the moral is "to be up and doing," so "that each tomorrow finds us farther than today," and by deed enhance this increasingly venerable profession.

With the premise laid and George prepared to go under the knife, there were two vital factors to be considered. First, the anesthesia. Ether was ruled out because of its exciting and suffocating tendencies, as was clearly demonstrated by but a few inhalations, which all but strangled the patient, when its use was attempted to produce quietude for the assurance of well-defined radiographs. Nembutal (pentobarbital sodium, Abbott), with the advantages of requiring little or no thought after its administration, was chosen and it served the purpose nobly. A narcotic dose was given orally, in capsules, two hours prior to the time set for operation, with the result that the patient went peacefully to sleep with the greatest of ease and without the slightest embarrassment of any of the vital functions. Immediately preparatory to the surgical treatment, a complementary intravenous injection was administered, to raise the dose to that usual for anesthetic requirements, namely, to a total of one-sixth of a grain for each pound of body weight.

The second important step was to provide artificial respiration during the period in which the lungs would remain in collapse, because of the atmospheric pressure vented into the thorax upon incision. This was accomplished through the aid of an ordinary automobile tire-pump, operated by an attendant at an approximate rate of 30 impulses per minute—not sufficiently forceful to inflate the lungs fully, yet enough to insure sufficient ventilation to sustain life. Obviously enough, no measure of the air intake was possible with such an arrangement, but it was vari-

ably provided by accelerating and retarding the pumping rate, as the condition of the patient demanded. Contact with the subject was made through the medium of a ⅜-inch standard rubber tubing inserted directly into the trachea by way of the mouth.

With everything in readiness, a liberal incision was made somewhat to the right of and parallel to the linea alba, commencing posterior to the xiphoid cartilage. A second was drawn at right angles to it, as in the formation of the capital letter T, into the right flank, thus providing a triangular opening for free access to the field of operation. An interior inspection, at once, revealed that the entire liver and an unmeasured section of small intestine were missing from the abdominal cavity and had prolapsed into the thoracic cavity through a rather irregular three-inch tear in the diaphragm, extending in a general dorso-ventral direction, parallel to and within an inch of its marginal attachment. The size and nature of the vent was, of course, visible only after the extrication of the abdominal organs which, to be sure, was a delicate operation and by no means as easily consummated as described. Gentle but rather firm traction, aided by the influence of gravity, afforded by the elevation of the fore part of the body, made possible the successful transposition. The subject was maintained in this inclined position until the conclusion of the correction to keep the abdominal viscera, now wrapped in antiseptic gauze, away from the surgical field, to facilitate subsequent suturing. Several ounces of a serosanguineous fluid and a large blood clot escaped from the thorax after the removal of the liver but otherwise the thoracic cavity and its contents appeared normal. The liver was decidedly swollen and passively congested.

The hernial ring was closed by two overlapping rows of continuous sutures, using Reeve's dry sterilized No. 2 catgut (15-day), each commencing at opposite ends of the aperture and there knotted with an allowance of a free end for subsequent tying to the terminal endings of the completed apposing series. The ascending series of stitches, the second to be inserted, interposed those of the descending chain, which gave the end result the appearance of a series of cross-stitches. Synchronous with the completion of the first row of stitches, the residual air in the thoracic cavity was expressed by a complete inflation of the lungs. The abdominal organs were now placed in approximate situ and the abdominal incisions were closed in the conventional manner, which was difficult because of the apposing influences

exerted by the swollen viscera and the pre-operative abdominal contracture. As this course progressed, it was both interesting and inspiring to observe the gradually increasing return of normal respiration, aided only by a moderate ventilation of the lungs with pure oxygen, as the impulses of air under pressure became unnecessary.

FIG. 1 (above). Reproduction of pre-operative x-ray negative, with bismuth meal. Stomach clearly shown *in situ* and intestinal loops in thorax.

FIG. 2 (below). Reproduction of post-operative x-ray negative, without meal, two weeks after operation.

Throughout the hour of operation, warmth for the patient and light for the operator were provided by reflected rays from ordinary incandescent electric bulbs and, of course, the usual asepsis was observed. As is unusual after the use of nembutal as an anesthetic, the patient recovered very calmly from under its influence. As to whether the divided means of dosage had anything to do with the incident is yet to be investigated. The after-treatment consisted only of quiet and the usual dietary regulation.

In conclusion, it may be remarked that 13 must be George's lucky number, for it was on July 13 that he underwent this noteworthy ordeal in quest of a normal existence, and it was on August 13 that he was dismissed from the hospital, completely restored. Thus, at this writing, three weeks since his departure, George continues to remain as hale and hearty as he ever was, enjoying the invigorating sea-breezes of his home surroundings and in the enviable position of commanding undivided and undisputed distinction whenever "my operation" is the theme of conversation. Needless to say, his mistress is very happy and possessed of a high esteem for the profession that made possible this auspicious reunion.

AN UNUSUAL OCCURRENCE OF CORNEAL DERMATOMA IN NEWBORN LAMBS*

By C. L. DAVIS, *Denver Colo.*
Branch Pathological Laboratory
U. S. Bureau of Animal Industry

Congenital malformations of the eye in newborn animals are seen occasionally in dogs and calves. These congenital anomalies have been seen also in horses, sheep and swine. Sporadic cases are reported in veterinary literature. Recently, Dr. J. C. Walker, of Holden, Missouri, reported a case in a newborn calf which showed a growth of normal hair on the eye-ball. The occurrence of this condition in numbers assuming epizoötic proportions is indeed unusual. Such an incidence was reported to our laboratory by Dr. W. T. Huffman, B. A. I. veterinarian stationed at Salt Lake City, Utah.

Subject: A recently born lamb.

History: About one-half of the first 225 lambs born in a certain band of sheep this spring were born blind. The affected

*Received for publication, October 8, 1934.

lambs showed deformed eyes with a growth on the surface of the eye-balls. Many of the growths contained tufts of hair although this feature was not constant (fig. 1). The affected lambs either were killed or died of starvation, with the exception of a few that were born dead. The same ewes and bucks running on the same range a year ago did not produce offspring showing this affection. There was no clinical evidence of infection in the ewes or bucks.

FIG. 1. Eyes from a newborn lamb showing corneal growths. Note the tuft of hair in one.

Histopathology: Sections prepared from the growth on the eye show structures which to a certain extent simulate those seen in normal skin and consist of a surface layer of flattened polygonal cells (*stratum granulosum*), several layers of large polygonal cells (*stratum germinativum*) composing the epidermis, and the deeper fibrous structure (the dermis), in which are seen cross-sections of hair follicles, blood-vessels and sebaceous glands (figs. 2 and 3). Pigment cells are present in the deeper layers of the epidermis. The growth rests upon the *substantia propria* of the cornea. This histologic picture is characteristic of dermoid growths.

DISCUSSION

The occurrence of an occasional congenital anomaly in an individual animal is ordinarily of little consequence. However, a high incidence such as reported in this instance is of considerable economic importance. One naturally wonders what factors might be responsible for such a condition. An attempt to offer an explanation of the etiology is only a conjecture. The mode of

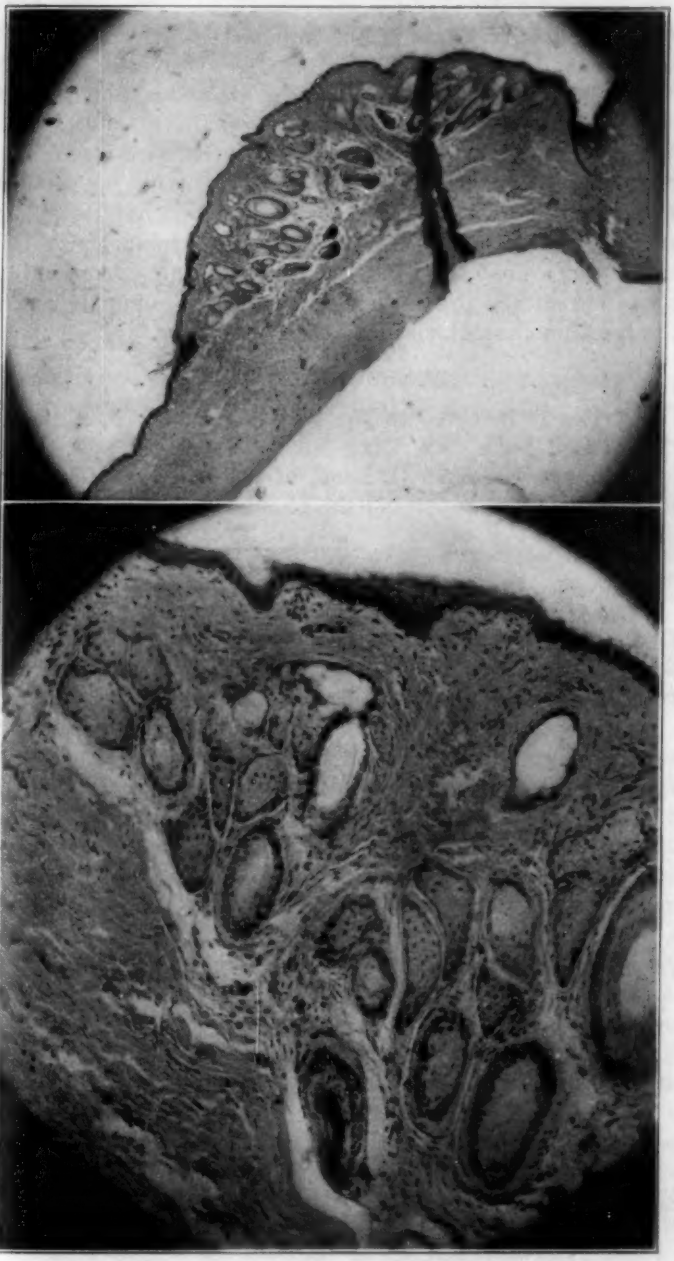

FIG. 2 (above). Section of the corneal growth (x 25).
FIG. 3 (below). Section of the corneal growth (x 125).

origin of congenital cysts is but imperfectly understood. These cysts, present in tissues where epithelial structures such as the skin and the cornea are normally seen, are probably formed from an embryonal displacement of the epidermal elements. It seems reasonable to exclude infection as a causative factor since an infectious process, in all probabilities, would result in some abortions. There is no history of abortions in this band of sheep although several were born dead. A possible explanation might be that the bucks used for breeding purposes came from a lineage that has hereditary predisposition to such an anomaly and have implanted this characteristic to their offspring.

ACKNOWLEDGMENTS

The writer is indebted to Dr. W. T. Huffman, B. A. I. veterinarian of Salt Lake City, Utah, for supplying the specimens and history, and to Dr. John C. Long, of the University of Colorado Medical School, for supplying the photographs.

Horse and Mule Business Recovering

That the horse and mule business is gaining steadily is the optimistic keynote of a letter sent out recently by Wayne Dinsmore, secretary of the Horse and Mule Association of America. Mr. Dinsmore points out that fairs had strong exhibits in all clases of horses and mules, and mentions especially the Ohio, Indiana, Illinois and Iowa state fairs as being particularly good, with huge crowds watching the horse shows.

Buying is on the increase, according to dealers in commercial horses and mules. They report that the demand for foals, yearlings and two-year-olds, and for mares from three to ten years of age, is so strong that prices are higher than they were last spring. Farmers are buying anything that looks as if it promises to grow into money. They are paying more now for two-year-old colts, than for mature horses ready to work.

Mr. Dinsmore goes on to say that tractor manufacturers claim their sales are increasing because of the rising prices of horses and mules, grain and hay, and are apparently planning a power sales campaign on that basis. To meet this competition, he asks the support of all who are interested in continued growth of the horse and mule business.

Beekeeping and honey production in Saskatchewan have been increasing at the rate of 10 per cent a year, it is reported.

PRÉCIS DE DROIT VÉTÉRINAIRE (Précis of Veterinary Law). Professor E. Liégeois, State Veterinary College, Cureghem-Brussels, Belgium. 331 pages. J. Duculot, Gembloux, Belgium. Price, 50 Fr.

The veterinarian should not confine his knowledge purely to the medical, surgical and zoötechnical phases. His activities are governed by laws which he must not disregard at the risk of becoming involved with the law. Furthermore, frequently he is called upon in the capacity of expert to place his knowledge at the disposal of legal authorities. Lastly, it is advantageous for him to be able to counsel a client on any occasion when the latter feels that he has been wronged.

The practicing veterinarian often becomes involved in matters which call for considerable knowledge of a variety of laws and regulations. With the exception of those that deal with sanitary police and meat inspection, these laws have been collected and divided into four parts in the "Précis of Veterinary Law."

By way of introduction and in order to give the reader some idea of legal phraseology, the author first deals with fundamental features of the law, especially those which have to do with giving expert testimony.

The part which deals with laws of business is arranged in such a way that one may easily find his way through the maze of laws and regulations relating to transactions involving animals. The author has reviewed decisions made by Belgian and French courts and gives the consensus of legal opinion prevailing in this type of case.

The concluding part of the book is devoted to professional laws governing not only teaching but the practice of veterinary medicine and pharmacy. Included in this part are laws governing official veterinarians.

As an appendix, the author presents specimens of legal documents which the veterinarian may be called upon to furnish from time to time. This book will be of particular interest to veterinarians who desire to familiarize themselves with the legal phases of the practice of veterinary medicine. The book is de-

signed to conserve the time of anyone who needs concise, authoritative information on the subject.

PUBLICATIONS RECEIVED

Bureau of Science, Philippine Islands, Thirty-first Annual Report of the. William H. Brown, Director. (Manila, P. I., 1933. pp. 869-952.)

Distribution of Mottled Enamel in the United States. H. Trendley Dean. Reprint No. 1581 from *Pub. Health Rpts.*, xlviii (1933), 25, pp. 703-734. Illus.

Estimation of Fluorides in Waters. Elias Elvove. Reprint No. 1596 from *Pub. Health Rpts.*, xlviii (1933), 40, pp. 1219-1222.

Studies on Bovine Mastitis. VIII. The Control of Chronic Streptococcus Mastitis. F. C. Minett, A. W. Stableforth and S. J. Edwards. Reprint from *Jour. Comp. Path. & Therap.*, xlvi (1933), 3, pp. 131-138.

Studies on Bovine Mastitis. IX. A Selective Medium for the Diagnosis of Streptococcus Mastitis. S. J. Edwards. *Jour. Comp. Path. & Therap.*, xlvi (1933), 4, pp. 211-217. Illus.

Studies on Bovine Mastitis. X. The Value of Field and Laboratory Tests for the Diagnosis of Chronic Streptococcus Mastitis. S. J. Edwards. *Jour. Comp. Path. & Therap.*, xlvii (1934), 1, pp. 49-60.

Infectious Bovine Mastitis. 2. The Streptococci of Chronic Bovine Mastitis. Wayne N. Plastridge, E. O. Anderson, G. D. Brigham and E. H. Spaulding. (Bul. 195. Storrs Agr. Exp. Sta., March, 1934. pp. 19.)

Field Observations on Weather Stain and Blowfly Strike of Sheep with Special Reference to Body Strike. F. G. Holdaway and C. R. Mulhearn. (Pamph. 48, Coun. Sci. & Ind. Res., Commonwealth of Australia, Melbourne, 1933. pp. 35. Illus.)

A Comparative Study of *Lolium perenne* and *Phalaris tuberosa* at Varying Stages of Growth. A. B. Cashmore. (Bul. 81, Coun. Sci. & Ind. Res., Commonwealth of Australia, Melbourne, 1934. pp. 32. Illus.)

The Insect Inhabitants of Carrion: A Study in Animal Ecology. Mary E. Fuller. (Bul. 82, Coun. Sci. & Ind. Res., Commonwealth of Australia, Melbourne, 1934. pp. 63. Illus.)

The Mortality Problem in Poultry. Morley A. Jull. (Inst. Amer. Poultry Ind., Chicago, Ill., 1934. pp. 11.)

Diseases of Sheep. J. A. Howarth. (Cir. 86. Calif. Agr. Ext. Service, August, 1934. pp. 71. Illus.)

Australian Veterinary Association

The Australian Veterinary Association will hold its annual conference at the Veterinary Research Institute, Parkville, Melbourne, January 16-23, 1935, during the centenary celebrations to be held in Melbourne at that time. The president and members of the Association extend to any members of the A. V. M. A., who may be visiting Australia during those dates, a cordial welcome to attend the conference.

STUDIES ON BOVINE MASTITIS. IX. A selective medium for the diagnosis of streptococcus mastitis. S. J. Edwards. Jour. Comp. Path. & Therap., xlvi (1933), 4, pp. 211-217.

Advantage was taken of the fact that streptococci are more resistant to the bactericidal action of crystal violet than the majority of milk saprophytes, and a selective medium has been devised which has afforded satisfactory results in the cultural diagnosis of bovine mastitis. Organisms which are not inhibited by the dye can be differentiated in blood-agar from mastitis streptococci by the inclusion of aesculin, upon which they act to produce black colonies.

S. J. E.

STUDIES ON BOVINE MASTITIS. X. The value of field and laboratory tests for the diagnosis of chronic streptococcus mastitis. Jour. Comp. Path. & Therap., xlvii (1934), 1, pp. 49-60.

(1). To obtain further experience of the relative values of field and laboratory tests for the diagnosis of chronic streptococcus mastitis, 809 cows in 18 herds were examined. Of these, 295 (36.4 per cent) were infected in at least one quarter with mastitis group I streptococci (*Str. agalactiae*). The field tests included the history of the cow, a test of milk of individual quarters for its reaction and the presence of clots. In the laboratory, the amount of deposit obtained by centrifuging was noted and a cultural examination made in or on a highly selective blood-agar medium, this being the basis of comparison. The value of cultural examination of mixed samples from the four quarters also was tested.

(2). Of 528 infected quarters of cows said to be *in full milk*, 62 per cent showed abnormalities to one or other of the indirect tests, but by the same criteria 9.4 per cent of milk samples from 2,284 healthy quarters also showed abnormalities. To avoid misleading results with quarters free from *Str. agalactiae*, it is preferable to accept as evidence of this infection only instances where both reaction and deposit tests are positive. When clots

are present in the fore-milk the cow may be regarded as infected, and this, when taken in conjunction with positive results to both reaction and deposit tests, is capable of revealing 41 per cent of cows infected with latent mastitis. In cows in the *drying stage*, cultural examination alone is of value.

(3). With 219 infected cows, examination of the mixed milk samples by deep plating showed the presence of streptococci in 91 per cent. The method was liable to fail only when non-hemolytic forms were present, usually in one quarter, or when the cow was infected with small numbers of beta-hemolytic streptococci. In 83 per cent of infected quarters, streptococci could be detected by surface cultivation.

(4). Procedures are outlined for the diagnosis of the disease in practice. The first plan aims at a quick and final diagnosis by cultural examination, while the second embraces a more extended application of field tests. It should be emphasized, however, that simple field tests can serve only for a preliminary survey of the herd, and the full extent of the disease can be determined only by a cultural examination of milk from individual cows.

S. J. E.

MORSURES VENIMEUSES MORTALLES CHEZ LES PORC (Fatal Snake Bites in Swine). A. Henry. Rec. de Méd. Vét., cx (1934), 3, pp. 129-131.

It is known that certain mammifers have a certain degree of natural immunity to the bite of venomous serpents. Swine are frequently cited as being refractory, as they are known to hunt for and to devour snakes. Where hogs roam they prevent the multiplication of snakes to a considerable extent.

The immunity of swine, according to various authorities, is attributed to three factors: first, the thickness of their skin, which prevents penetration of the serpent's fangs; second, the low vascularity of the subcutaneous fat, which prevents absorption and perfusion of the venom and, third, a certain antitoxic action of swine blood on snake venom. From investigations made by Calmette in Indo-China, the low vascularity of the subcutaneous fat is the main protective factor. V. Brazil (1911) asserted that the wild hog of Sao Paulo is immune, as they search for snakes and eat them as nourishment, although some of the immunity, at least, is lost on domestication. Bullard (1910) demonstrated with guinea pigs that the autolysed liver juice of hogs had antitoxic action against viper venom.

The author could not confirm the presence of natural immunity of swine. A mixture of equal parts of blood of the domestic hog and venom was not antitoxic for rabbits, and he reports the death of two hogs from the bite of vipers. These swine were housed in a pen adjacent to a stone quarry inhabited by vipers which were attracted to the pens by the skim milk fed to the hogs. Two hogs bitten on the thin skin of the thigh died of typical snake-bite toxemia. The conclusion is that swine are not really immune to the bite of venomous serpents.

L. A. M.

REMEDIES FOR CYANIDE POISONING IN SHEEP AND CATTLE. A. B. Clawson, H. Bunyea and J. F. Couch. Jour. Wash. Acad. Sci., xxiv (Sept. 15, 1934), pp. 369-385.

The authors tested the efficiency of four suggested remedies for hydrocyanic acid poisoning: sodium thiosulfate, sodium nitrite, sodium tetrathionate and methylene blue. A combination of nitrite and thiosulfate was used in the cattle series.

The animals used were sheep and cattle. The poison was administered as potassium cyanide and was given by drench. The remedies were given intraperitoneally to the sheep and intravenously to the cattle.

As a preliminary measure the m. l. d. and m. t. d. of hydrocyanic acid, given as potassium cyanide by mouth, was determined. For sheep, the m. l. d. was determined to be 2.315 mg per kilo calculated as HCN; for cattle, nearly 2.042 mg. The m. t. d. was found to be, for sheep, 0.992 mg per kilo and for cattle somewhat less than 0.882 mg.

In the experimental work with sheep, 50 cc of methylene blue protected against 1.42 lethal doses of cyanide, although 30 cc failed to do so. Of the other remedies tried, sodium tetrathionate and sodium nitrite, each protected against 1.42 m. l. d. but failed to do so against slightly larger doses. Sodium thiosulfate protected against 1.62 m. l. d. The combination of sodium thiosulfate and sodium nitrite was not tried with sheep.

In the cattle experiments, sodium nitrite protected against a single m. l. d. of cyanide but failed to do so when 1.5 m. l. d. had been given. Sodium thiosulfate protected against 1.39 m. l. d. With cattle the best results were obtained with a combination of sodium nitrite and sodium thiosulfate which protected against 2 m. l. d. Methylene blue and sodium tetrathionate were not used with poisoned cattle.

The results strongly indicate that in administering any of the substances tried as remedies it is of the utmost importance that they be given very promptly after symptoms of poisoning develop and before the period of respiratory paralysis sets in.

Aside from these remedies no other treatment was given the sick animals. It is the authors' opinion, based on their observations of the course of the sickness, that in several cases additional treatment such as the stimulation of respiration and general supportive measures would possibly have altered the final result of the cases. It is suggested that, in cases of cyanide poisoning, treatment with the remedies used in this study could well be supplemented by other measures with better chances of success.

Two experiments indicate that the remedial action of nitrite and tetrathionate is physiological rather than chemical.

H. W. S.

THE LIVER FLUKE (FASCIOLA HEPATICA) IN FRANCONIA. Liver fluke disease in the region of the river Regnitz and the adjoining valleys on its left bank from Fürth to Bamberg and the spread of the snail host, *Galba trunculata*. Samuel Mehl. Landwirtschaftlichen Jahrbuch für Bayern (1931), 10 and 11. (Carl Gerber, Munich, Germany.)

The author, after discussing the life history of the liver fluke, the discussion being based in part on his own observations, and after pointing out several errors concerning certain phases of this life history, which, he states, have crept into the literature, studies and correlates the ecological conditions favoring the existence and spread of the intermediate snail host, the weather conditions and the prevailing agricultural practices with the occurrence of outbreaks of liver-fluke disease and their severity in the area under consideration, and outlines preventive measures based upon these studies and correlations.

While the information contained in Dr. Mehl's paper is chiefly of local interest, it may be applied to other areas in which similar conditions prevail. The paper shows that a thorough knowledge of the ecological and climatic conditions which favor the maintenance and spread of the intermediate host of the liver fluke is of primary importance in any area where liver-fluke disease is a problem and that, armed with this knowledge, it is often possible to devise preventive measures by changing natural conditions in such a way as to render them unfavorable for the continued existence of the snail host.

Dr. Mehl's thorough work again demonstrates that, granted a knowledge of the underlying causes, there is nothing mysterious in apparently sudden outbreaks of liver-fluke disease. The paper is a very useful addition to our knowledge of this trouble.

G. D.

PROPHYLAXIE DE LA DYSENTERIE DES VEAUX (Prophylaxis of Calf Scours). J. Basset. Rev. Vét & Jour. de Méd. Vét. et de Zoot., lxxxvi (March, 1934), pp. 129-134.

Coli-bacillary septicemia, or dysentery of calves, is well known since the time of Jensen (1893). It appears a few hours after birth and often before the first sucking, ends mortally in from two to four days and in certain years kills from 30 to 40 per cent of all the newborn of some herds. The dehydrating diarrhea reduces the victims to mere skeletons. The intestinal contents show immense quantities of bacteria mixed with numerous epithelial cells, grouped or isolated, which have been mistaken for protozoa. Bacteriological analysis, however, shows the disease to be a coli-bacillary septicemia. In one of three cases, examined 18 hours after death, an enterococcus was found associated with *B. coli*, five strains of which have been identified by Weinberg and Prévot. They are differentiated from paratyphoid organisms by their behavior in sour milk media.

The early appearance of the symptoms, the rapid evolution of the disease and the multiplication of an organism of relatively feeble virulence lead to the conviction that calf scours is a transplacental infection. The fetus does not die before birth because it lives under more favorable conditions in the uterus, as does the chick infected with fowl typhoid before hatching. The cow is no more harmed by *B. coli* than is the chronic carrier (hen) of the paratyphoid organisms. The brutal revolution of the newborn life rapidly transforms a latent into a septicemic infection.

These facts have pointed out to various authors that mothers of the different species of animals should be vaccinated against the organisms habitually responsible for septicemias of the newborn. Vaccines (bacterins) made from colon bacilli isolated from calves affected with dysentery for this observation were injected at 10- to 15-day intervals during the month preceding parturition. The results were not decisive, as was shown by vaccinating half of the cows in the herds and retaining the others as checks.

In regard to the success of treatment, the author indicates that the early appearance of the disease occurs from a copious intra-

uterine infection. Cases occurring after the fourth day yield to the usual treatment, or lead to arthritis, suppuration, and death later on.

The important thing in calf scours is to "rehydrate" the body, as is done with patients (human) affected with cholera. Intravenous injections of hypertonic salt solution (2 liters at 1.2 per cent) every 24 hours and a hydrating diet (rice water and legume teas) are recommended, after which one can always offer the patient a dose of bacteriophage (super) and a "bouquet of vitamins," together with injections of the mother's blood. At least, these are the steps one is supposed to take.

Concerning the prophylaxis, control cannot be based upon detecting latent infections among the adults as in the case of fowl typhoid, for example. *B. coli* is a permanent but undesirable parasite of the digestive tract ever capable of penetrating the tissues and provoking trouble, chronic and acute, under various conditions, among which are the arresting or slowing of peristalsis during the winter months in pregnant cows kept in more or less vitiated air under unfavorable hygienic conditions for five or more months and fed with dry feed, oil cake and ensilage. Winter breeding may be practiced for economic reasons, but the fact remains that calf scours is frequent in winter and rare in the summer. As cows never become immune to *B. coli*, which causes so much trouble, the author thinks the present breeding custom should be changed. Calving after the exercise, the sunlight, the open air, and the stimulating action on the digestive tube, incident to pasturing, would have a wholesome effect on the occurrence of this fatal disease of the newborn. In lieu of such a radical change, outdoor exercise and feeding such as would effect a transition of plethoric alimentation are the preventive measures to employ and these are feasible.

L. A. M.

Did You Get Your Copy?

If you have not received a copy of a recent issue of the *Bloodless Phlebotomist*, published by the Denver Chemical Manufacturing Company, of New York, you should write and ask for a copy of number 2 of volume VIII. The publisher recently mailed a copy of this issue to every known physician and veterinarian in the United States. In addition, this little journal has been translated and printed in nine languages, and a copy mailed to every doctor in the world with a known address, excepting in the countries of Russia, Latvia and Bulgaria. The various editions total 1,347,000 copies.

Regular Army

Major Lloyd C. Ewen is relieved from his present assignment and duty at Fort Sam Houston, Tex., effective at such time as will enable him to proceed to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about October 13, 1934, for the Hawaiian Department.

Lt. Col. Clell B. Perkins is, in addition to his other duties, detailed as attending veterinarian at Forts Slocum and Wood, New York, the harbor defenses of eastern and southern New York and Sandy Hook, N. J., and at the headquarters, Second Corps Area.

The following officers in the Veterinary Corps, having been found by an Army retiring board incapacitated for active service on account of disability incident thereto, and such finding having been approved by the President, their retirement from active service on September 30, 1934, under the provisions of section 1251, Revised Statutes, and the act of Congress approved April 23, 1930, is announced:

Colonel Walter Fraser
Colonel William A. Sproule
Colonel Andrew E. Donovan
Colonel Robert C. Musser
Major Vincent B. Wright

Veterinary Reserve Corps

New Acceptances

Johnston, Elmer Jos.....Major...Box 730, Excelsior Springs, Mo.
Anderes, Robert Louis....2nd Lt..1817 Church St., Evanston, Ill.
Bailey, Lee Kenneth.....2nd Lt..Leesburg, Va.
Bentham, Wilfred Sylvester
.....2nd Lt..5430 Claremont Ave., Oakland, Calif.
Ellis, Jos. Leslie.....2nd Lt..5214 S. Sheridan Ave., Tacoma, Wash.
Merriman, Clarence Chas.2nd Lt..5618 S. 19th St., Omaha, Neb.
Purse, Wm. Jos.....2nd Lt..9357 E. Colfax Ave., Aurora, Colo.
Smith, Robt. Markwood..2nd Lt..W. Washington St., Jamestown, Ohio.

Promotions

To

Hart, Charles Henry.....Major..138 King St., Saint Paul, Minn.
Sanders, Charles Lee.....1st Lt..705 S. Main St., Dayton, Ohio.

Transferred to Aux. Reserve

Turner, John Pollard.....Col.....1357 Kennedy St., N. W., Washington, D. C.
McKibbin, Robert Webber.Capt....216 W. 2nd St., Waynesboro, Pa.

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Foreign Membership

Final figures on the enrollment of members from foreign countries, in the Twelfth International Veterinary Congress, show that 61 countries contributed 755 members. Germany heads the list with 133, followed by Great Britain with 97. Canada is credited with 52 and Belgium with 44. Yugoslavia, with 38; France, with 31; Czechoslovakia, with 30; Chile and Roumania, each with 28, follow in the order named. The complete list follows:

Algeria	2	Japan	14
Argentina	2	Kenya	1
Australia	14	Latvia	2
Austria	6	Lithuania	1
Belgium	44	Manchuria	1
Bermuda	1	Mexico	5
Brazil	13	Morocco (French)	2
Bulgaria	2	Morocco (Spanish)	1
Canada	52	Netherlands	18
Chile	28	Netherlands East Indies	13
China	3	New Zealand	2
Cuba	14	Nigeria	2
Cyprus	1	Norway	5
Czechoslovakia	30	Palestine	1
Denmark	4	Peru	6
Dominican Republic	1	Poland	7
Ecuador	1	Portugal	6
Egypt	14	Roumania	28
Estonia	1	Spain	17
Finland	4	Straits Settlements	1
France	31	Sweden	9
Germany	133	Switzerland	14
Great Britain	97	Tanganyika Territory	1
Greece	2	Trinidad and Tobago	2
Hong Kong	1	Tunis	1
Hungary	11	Uganda	2
India	1	Union of South Africa	10
Indo-China	1	U. S. S. R.	3
Irish Free State	7	Uruguay	3
Italy	17	Yugoslavia	38
Jamaica	1		
		Total	755

Educational Exhibit at the I. V. C.

A feature which added to the completeness of the Twelfth International Veterinary Congress was an extensive educational exhibit. It occupied the entire floor space of the luxurious

Basildon Room, which measures 37 by 48 feet. The exhibit consisted of six principal booths and numerous supplementary charts, maps, preserved specimens, and scientific apparatus.

Service of the Bureau of Animal Industry

One display, which was prepared by the U. S. Department of Agriculture especially for the Congress, was entitled "Services of the Bureau of Animal Industry of the U. S. Department of Agriculture." The presentation consisted of a live stock scene mounted on a series of foundation stones representing veterinary science, improved breeding, and sound feeding and management —all being presented as a substantial foundation for the development of stock-raising in the United States. Realistic cut-out figures of the principal species of domestic live stock, including poultry, were accompanied with statistics showing the number of each kind of animal in the country.

A panel at the right of this display portrayed, by illustrations and text, important regulatory measures conducted by the Bureau to safeguard the live stock industry. The left-hand panel displayed important results of scientific investigation, illustrating the research phase of the Bureau's contributions to stock-raising and public welfare. Various tests, biological products, disinfectants, and other specific results of federal research work constituted a portion of the display.

A further part of the presentation was a stereopticon machine which showed continuously a series of 70 photographs and supplementary titles portraying the Bureau's services.

The Egg Factory

An ingenious mechanical display entitled "The Egg Factory" but renamed "The Talking Hen" by many visitors, showed the various biological operations in the production of eggs. This exhibit, measuring about eight feet square, illustrated the sequence of operations from the beginning of the egg in the ovary to the final product wrapped by nature in the shell. Through a loud speaker concealed in the exhibit, a phonograph record told the story of egg-production with special reference to proper feeding and care.

Advice from an Old Ewe

Methods of preventing and controlling stomach worms were presented similarly, by phonographic reproduction, in an exhibit of lifesize models of a ewe and her lamb. Speaking in the first person, the ewe gave advice to sheep-owners on ways to increase their returns from sheep-production by a more effective control

of parasites, particularly stomach worms. A pasture setting added to the realistic character of this display.

Research Conquers Cattle Ticks

In another mechanical exhibit the life cycle of cattle ticks and the effectiveness of systematic dipping in breaking the cycle and eradicating ticks were demonstrated. Two large revolving disks, carrying models of seed ticks and mature ticks, respectively, acquainted the observer with essential information concerning the appearance and habits of this pest of the cattle industry in the South.

Immunizing Suckling Pigs Against Hog Cholera

The feasibility of protecting suckling pigs against hog cholera by immunization with serum and virus was demonstrated in a display which presented the results of experimental work in this important veterinary field. The exhibit showed the economy of immunizing pigs while still of suckling age and also the effectiveness of the treatment in creating lifetime immunity.

Parasite Strategy Charts

Novel means of organizing veterinary attacks on animal parasites were presented in a series of five panels containing numerous charts. Each chart showed a type of military strategy suitable for combating a particular kind of parasite enemy. The purpose of this display was to show the best opportunities for attacking parasites at vulnerable points in their life cycles.

Other Exhibits

An adjoining panel contained recently prepared organization charts of the federal Bureau of Animal Industry which illustrated the scope of activities conducted by its twelve principal divisions. This display was of particular interest to foreign visitors charged with conducting veterinary organizations. Numerous maps and charts illustrated also the excellent progress of veterinary campaigns against bovine tuberculosis, tick fever, and other animal maladies. Several racks of preserved specimens, showing lesions and abnormal conditions found on postmortem examination, lent diversity to the exhibit.

Another small exhibit dealt with controlling roundworms of hogs and contrasted the effects of insanitary hog lots with the advantageous use of clean pastures.

The foregoing educational displays were prepared and exhibited by the U. S. Department of Agriculture as a memorial to 50 years

of service to the live stock industry and the public since the Bureau was created in 1884. In addition, the Mayo Foundation presented in the same room an exhibit illustrating various pathological conditions of domestic animals.

The exhibit room was located conveniently between the registration desk and the various rooms and foyers where general and sectional meetings were held. As a consequence, the educational exhibits were viewed by the great majority of delegates and visitors to the congress.

D. S. B.

Congress Proceedings

Letters are being received from members of the Twelfth International Veterinary Congress, inquiring about the Proceedings. The By-laws of the International Veterinary Congresses specify that the proceedings of each Congress shall be published within one year of the closing date of the Congress. The publication of the Proceedings of the Twelfth Congress will entail more work than has been the case with any previous meeting, for several reasons. The number of reports to be published is larger than for any previous Congress, and the addition of Spanish as an official language also adds to the amount of material to be edited and published. The compilation of the lists of various classes of members, totaling approximately 3800, is no small task in itself. It is estimated that the work of editing and printing will be completed within the time specified, and it is possible that members of the Congress will receive their copies of the Proceedings within eight or ten months after the Congress.

Mare Adopts Two Calves

Mr. John Kernohan, M. R. C. V. S., of Ballymena, England, reports an interesting case of a mare belonging to a client of his that is suckling two young calves, according to the *Veterinary Record*. Mr. Kernohan records his observations as follows:

> Eight calves were grazing in the same field with the mare, which is nine and one-half years old, and 15.1 hands high. The evening drink was brought for the calves, and a red calf, as soon as it had finished, went over to the mare and began suckling. A few seconds later, a roan calf followed. Both drew from the mare at the same time. A remarkable feature of this case is that the mare has never had a foal, or ever been served, yet she has plenty of milk.

Compulsory school attendance ages vary in the different states. No state requires children to start school before the age of six.

NEW YORK UNIVERSITY ALUMNI ASSOCIATION

The Veterinary Alumni Association of New York University held a dinner meeting at the Waldorf-Astoria Hotel, New York City, Tuesday evening, August 14, 1934. The Association is made up of graduates of the American Veterinary College, the New York College of Veterinary Surgeons, the New York-American Veterinary College and the New York State Veterinary College of New York University.

After an excellent dinner, the acting Chairman, presiding in the absence of the President, Dr. A. N. Towner, called on Dr. Lester H. Howard, of Boston, Mass., of the class of '82, the senior member present, for a few remarks. Dr. Howard responded, expressing his pleasure at being able to attend another alumni gathering. The Chairman then called on a number of other members, in order of their seniority. Dr. R. J. Garbutt, of the class of '21, responded as the youngest member present.

The reunion of three members of the class of '94, Drs. John C. Petersen, Alfred J. Tuxill and R. C. Gross, who had never met since they were graduated more than 40 years ago, was one of the happy events of the meeting.

A telegram expressing the sympathy of the Alumni Association and of the Veterinary Medical Association of New York City was sent to Dr. O. E. McKim, of Port Chester, N. Y., on the death of his father, which occurred during the sessions of the Congress. Telegrams of sympathy and flowers were sent to Dr. George H. Berns, of Brooklyn, N. Y., class of '79, and to Dr. James T. Glennon, of Newark, N. J., class of '96, who were unable to be present because of illness.

Thirty-five members and one guest were present, the largest attendance in many years. Those in attendance were: Drs. Lester H. Howard, '82; Frederick P. Ruhl, '85; W. F. Harrison, '87; W. H. Kelly, '89; Herman Kock, E. N. Stout, H. K. Miller, E. B. Ackerman, J. Payne Lowe, Charles E. Caulfield, all of '91; W. H. Dodge, '92; John C. Petersen, R. C. Gross, Alfred J. Tuxill, R. S. MacKellar, W. C. Siegmund, all of '94;

W. R. Rosekrans and L. D. Ives, '96; E. F. Sanford and Arthur W. Smith, '98; W. M. MacKellar, '99; John J. Hayes and A. Eichhorn, '00; R. G. Bose and Joseph L. Serling, '01; J. Elliott Crawford, '04; C. G. Rohrer and Charles S. Chase, '06; Oscar J. Kron, Edward L. Baldwin and Herbert L. Cox, '07; W. B. Maxson, '11; S. M. Apfelberg, '13; J. R. Porteus, '18; R. J. Garbutt, '21. The guest was John W. Kasten, a student in the School of Veterinary Medicine, University of Pennsylvania.

R. S. MACKELLAR, *Chairman.*

INTERSTATE VETERINARY MEDICAL ASSOCIATION

The twenty-first annual meeting of the Interstate Veterinary Medical Association was held at the Warrior Hotel, Sioux City, Iowa, October, 18-19, 1934. Although the registered attendance of 157 lacked three of equalling the all-time record set in 1933, the excellence of the program and the interest shown by those in attendance have never been exceeded.

Dr. A. L. Howard, of Sioux City, opened the program with a brief but timely address. Dr. W. L. Boyd, of the University of Minnesota, opened the literary program with a discussion of "Bang's Disease Control." In view of Dr. Boyd's long experience in pioneering in this field and due to the program that is being inaugurated by the federal government in the control of Bang's disease, his listeners were keenly interested. Many questions were asked. Another timely discussion was that on "Drouth Emergency Problems," by Dr. R. G. Elliott, of Aberdeen, S. Dak., who described some of the details of purchasing animals in the drouth-stricken area. Dr. A. D. Bullock, U. S. B. A. I., inspector-in-charge, Sioux City, followed Dr. Elliott and told how these animals are classified and distributed. He said that 330,000 cattle had been cleared through Sioux City and that most of them came from the Dakotas, Nebraska and states west. Approximately one-half of these were slaughtered in Sioux City. Nearly 30 per cent were shipped to states south of Kentucky and east of the Mississippi for grazing. The rest went east for slaughter.

Dr. Elmer Lash, of the U. S. B. A. I., Washington, D. C., who was present as a visitor, was called on and briefly discussed some phases of the federal program. Dr. A. A. Fosterman, of Utica, S. Dak., discussed some of the vexing problems of the sales pavilion. He recommended that all such pavilions in all states should be placed under uniform regulations to avoid some of the practices prevalent at the present time. Dr. W. L. Boyd was recalled to the floor to discuss "Diseases of Sheep." He also

touched on the diseases in cattle that have cropped up recently as the result of plant-poisoning and nutritional disturbances.

With the Association banquet in the evening, the activities of the first day were brought to a close. Dinner was served to 135 veterinarians and their friends at the Warrior Hotel. Dr. G. R. Fowler, of Iowa State College, was toastmaster. He reported on the Twelfth International Veterinary Congress held in New York City in August. His comments were interesting, with touches of humor that were thoroughly enjoyable. He called upon one representative from each of the four states present for a few brief remarks. Following the banquet, the members were guests of the commercial organizations at a dance.

First to appear on the second-day program was Dr. H. F. Beardsley, of Lakefield, Minn., who presented an exceptionally fine treatise on "Equine Encephalomyelitis." He reported on 75 cases seen in his practice since July, 1934, and illustrated his description with a pair of splendid moving picture reels taken of some of his patients. In this epizoötic, the disease seemed to spread by contact, and specific serum administered apparently gave good results. Next on the program was Dr. F. M. Wilson, of Mechanicsville, Iowa, who is well known as the organizer of the annual Eastern Iowa Practitioners' Clinic. Dr. Wilson gave an unusually excellent talk on "Treatment of Common Equine Ailments." The revived interest in horse practice was well illustrated by the close attention given to the discussion. Dr. G. R. Fowler described new features in large-animal surgery. Lantern slides were shown in connection with the talk. Dr. J. E. Weinman, of Lincoln, Neb., talked on small-animal practice and made many helpful suggestions on the treatment of common ailments.

Dr. C. E. Juhl, of Osage, Iowa, president of the Iowa Veterinary Medical Association, presented a splendid paper on "Factors in Swine Reduction," covering the field of swine diseases from inception to maturity. The program closed with a discussion of a new scourge of northwest Iowa, the Texas screw-worm. Mr. Knipling and Mr. Tate, of the Entomology Department of Iowa State College, gave the life history of the pest and the history of its invasion of northern points. This is the first time it has been found in Iowa.

Ladies who attended the Auxiliary meeting reported a very successful get-together.

Officers chosen for the coming year are: President, Dr. D. C. Scott, Tekamah, Neb.; vice-president, Dr. E. L. Eggleston, Alcester, S. Dak.; secretary-treasurer, Dr. W. A. Aitken, Merrill, Iowa (reëlected).

W. A. AITKEN, *Secretary.*

TRI-COUNTY VETERINARY ASSOCIATION

The monthly meeting of the Tri-County Veterinary Association was held at the Hotel Winona, Winona, Minn., October 25, 1934. The attendance of veterinarians and their wives was the largest in the history of the Association. Dr. John R. Mohler, chief of the U. S. Bureau of Animal Industry, was the guest of honor.

The program for the day was in charge of Dr. N. A. Roettiger, of Winona, and Dr. George S. Failing, of Lewiston. The subject, "Eradication of Bang's Disease," was discussed by four speakers: Drs. John R. Mohler, of Washington, D. C., W. J. Fretz, C. P. Fitch and W. L. Boyd, of Saint Paul. Dr. P. H. Radford, of Slayton, president of the Minnesota State Veterinary Medical Association, spoke on "Diseases of Swine," and Dr. H. J. Shore, of Fort Dodge, Iowa, talked on "Equine Encephalomyelitis."

The ladies were entertained by members of the local Auxiliary.

The event came to a successful close with the banquet in the evening, with Dr. H. C. H. Kernkamp, of Saint Paul, fulfilling the duties of toastmaster efficiently and delightfully.

P. H. RIEDE, *Secretary.*

Tuberculosis-Free States

In answer to several inquiries, and believing that the information might be useful to others, we are publishing a list of the states that have been designated as tuberculosis-free, with the date when each state acquired this distinction.

October 1, 1928	North Carolina
May 1, 1929	Maine
August 1, 1930	Michigan
July 1, 1931	Indiana
January 1, 1932	Wisconsin
	Ohio
June 1, 1932	Idaho
July 1, 1932	North Dakota
March 1, 1933	Nevada
July 1, 1933	New Hampshire
	Utah
November 1, 1933	Kentucky
December 1, 1933	West Virginia
May 1, 1934	Washington
September 1, 1934	Illinois

NECROLOGY

ARTHUR LAVERNE TIFFANY

Dr. A. L. Tiffany, of Monroe, Mich., died at the Ann Arbor Hospital, February 6, 1934, of heart disease. He had been ill for about six months.

Born in Pennsylvania, November 30, 1874, Dr. Tiffany was graduated from the Ontario Veterinary College in 1905. Immediately following his graduation, he located in Monroe and practiced there until his death. He was Monroe County Veterinarian for many years. In addition to his veterinary practice, he was also engaged in the real estate and insurance business.

Dr. Tiffany joined the A. V. M. A. in 1923. He was active in civic affairs, serving on both the school board and the city council. He was an active Mason, and had devoted much of his time to planning and financing the Monroe Masonic Temple. His funeral was held from the Temple.

S. G. C.

ERNEST LINWOOD CORNMAN

Dr. Ernest Linwood Cornman, of Marietta, Pa., died at his home, July 25, 1934, following a heart attack suffered while fishing one of his favorite streams the day before. Dr. Cornman had been in ill health since November 15, 1933, when he fell during the National Beagle Trials at Leesburg, Va., and had become active again only about ten days prior to his death.

Born in Montgomery County, Pa., July 16, 1875, Dr. Cornman received his preliminary education in the public schools of Montgomery County and in the Lower Merion High School, before entering the School of Veterinary Medicine at the University of Pennsylvania. Following his graduation in 1900, he practiced for seven years, first at York, Pa., and later at Gladwyne, in Montgomery County. In January, 1907, he became associated with the Dr. H. M. Alexander & Company Laboratories, Inc., at Marietta, Pa. In 1916, this Company became the Gilliland Laboratories, Inc., and Dr. Cornman continued with them, becoming President just prior to the depression. Through his energy and

wise direction, the Laboratories came through the economic depression in splendid shape.

Dr. Cornman joined the A. V. M. A. in 1907. He was a member of many fraternal and civic organizations: vice-president of the Lancaster Automobile Club; director of the Lancaster General Hospital; chairman of the Marietta Welfare Federation; a member of the Lancaster Kiwanis Club; the Lancaster Lodge of Elks; Ashara Lodge, No. 398, Thirty-Second Degree, F. and A. M.; Zembo Temple Shrine, of Harrisburg; the Lancaster Medical Club, the American Public Health Association, the Pennsylvania Public Health Association, Conestoga Veterinary Club, Sportsmen's Club of West Chester, and a number of beagle clubs. Surviving are his widow, two sons and a daughter.

E. K. T.

THERIES D. HINEBAUCH

Dr. Theries D. Hinebauch, of Long Beach, Calif., died August 6, 1934. He had been in failing health for several years.

Born at Vicksburg, Mich., July 15, 1860, Dr. Hinebauch was graduated from the Michigan Agricultural College in 1885, and from the Ontario Veterinary College in 1887. In January, 1888, he became a member of the staff of Purdue University, at LaFayette, Ind., where he remained three years. In 1891, he became the first Professor of Veterinary Science at the North Dakota Agricultural College, at Fargo. Subsequently, he was appointed the first State Veterinarian of North Dakota, under the law of 1895. He was a prolific writer on veterinary and agricultural subjects. He remained at Fargo until 1897, when he resigned to engage in practice at Tower City, N. Dak. In 1914, his failing health made it necessary for him to retire from active professional work and he moved to California where he had since made his home.

Dr. Hinebauch joined the A. V. M. A. in 1892 and resigned in 1917. He had been a member of the North Dakota Veterinary Association and was also an honorary member of the Illinois State Veterinary Medical Association. He is survived by his widow (née Priscilla Bowers).

L. M. R.

EDGAR SINCLAIR BROWNE

Dr. Edgar S. Browne, of Devonshire, Bermuda, died July 30, 1934, of cardiac complications. He was a graduate of the Ontario Veterinary College, class of 1923, and had practiced in

Toronto for a short time after graduation, before returning to Bermuda.

JOHN MICHAEL BAKER

Dr. John M. Baker, of Saint Walburg, Sask., died September 24, 1934, following an attack of infantile paralysis. Born at Estlin, Sask., December 8, 1908, Dr. Baker was graduated from the Ontario Veterinary College in 1932. After graduation, he located for general practice in Saint Walburg. Dr. Baker joined the A. V. M. A. in 1933. On July 5, 1934, he was married to Miss Irene Babineau, of Paradise Hill, Sask. Besides his widow, he leaves his parents and three brothers.

I. B. B.

JUDSON BLACK

Dr. Judson Black, of Lansing, Mich., formerly of Richmond, Mich., died at the home of his daughter in Detroit, Mich., September 28, 1934.

Born in Northwood, Ont., March 29, 1859, Dr. Black was graduated from the Ontario Veterinary College in 1894. He immediately became active in the affairs of the veterinary profession of Michigan, where he had located for practice. The proceedings of the Michigan State Veterinary Medical Association bear ample testimony to the work and worth of this stalwart of the profession. Until his declining years, he was practically always serving in some capacity for the Association. He was President in 1898 and again in 1912, and served as Secretary-Treasurer without interruption for the ten-year period, from 1903-1912. When it is realized that it was during this term of office that the present Veterinary Practice Act was passed and made effective, the size of the task can be appreciated.

Dr. Black joined the A. V. M. A. in 1903 and served as Resident Secretary for Michigan for one term (1907-08). He served officially as a member of the Veterinary Examining Board of Michigan, and when the Board was absorbed by the newly created Department of Agriculture in 1921, he was retained as superintendent of stallion registration, a position he held until he left the Department in 1933. He was a member of the Masonic Lodge and a devout church member.

Dr. Black was a courteous, genial gentleman of the old school, and will be greatly missed by the younger members of the profession as well as the old guard. Burial was made at Richmond,

Mich., beside his wife, who preceded him in death by less than a year. The couple had celebrated their golden wedding anniversary on Christmas Day, 1932. He is survived by a daughter and a granddaughter. B. J. K.

C. G. DEENIS

Dr. C. G. Deenis, of Ottawa, Ill., died at his home, September 29, 1934. He had been in ill health for several years.

Born in Rutland Township, Ill., October 5, 1859, Dr. Deenis was graduated from the Chicago Veterinary College with the class of 1893. Shortly thereafter he established an office in Ottawa where he practiced until his retirement in 1918. He early became known as one of the leaders of the veterinary profession in northern Illinois.

Dr. Deenis was a member of Occidental Lodge, No. 40, A. F. & A. M., and of the Ottawa Commandery, No. 10, Knights Templar. He was a member of the city council for twelve years and, for several years, served as city milk inspector. He leaves his widow (née Ella A. Curyea), a sister, two nieces and a nephew. He and Mrs. Deenis had celebrated their golden wedding, December 5, 1933.

FREDERICK P. RUHL

Dr. Frederick P. Ruhl, of Milford, Del., died suddenly at his home, October 4, 1934. He had been ill with heart disease for several months.

Born in New York, N. Y., December 25, 1866, Dr. Ruhl entered the American Veterinary College in 1883, at the age of 17, and was graduated two years later. Following his graduation, he located for practice in Yonkers, N. Y., and remained there about ten years. In 1895, he removed to Fairmont, W. Va., and, six months later, was appointed consulting veterinarian to the West Virginia State Board of Agriculture. Three years later, he was elected to the Chair of Veterinary Science at West Virginia University, and occupied himself with teaching for the next six years. In 1904, ill health forced him to relinquish this position. Being advised by physicians that he could not live more than a few years, he moved to a small farm near Milford, Del. After a year of farming, he had regained his health to such an extent that he re-entered active practice. When Dr. Ruhl retired, the latter part of 1933, he had been in practice for 49 years. It was a source of considerable regret to him that his health would not permit him to round out 50 years in his chosen profession. Dur-

ing recent winters, he had traveled quite a bit for the purpose of benefiting his health.

Dr. Ruhl joined the A. V. M. A. in 1900. He served as Resident Secretary of West Virginia (1900-01; 1902-05), and of Delaware (1919-21). He was a charter member of the old West Virginia State Veterinary Medical Association (reorganized in 1930), and also of the Delaware Veterinary Medical Association, which he served as President for one term. He is survived by his widow.

C. C. P.

LESTER B. RINEHART

Dr. Lester B. Rinehart, of Cooper Township, near Plainwell, Mich., died suddenly at his home, October 5, 1934. He had just returned from making a call when he was stricken with a heart attack and died before medical aid could be summoned. He was 58 years of age. A graduate of the Grand Rapids Veterinary College, class of 1905, Dr. Rinehart had practiced in Cooper since his graduation. Surviving are his widow, four sons, three daughters and one grandson.

FEDERICO DE GASPERI

Dr. Federico De Gasperi, Director and Professor of General Pathology and Pathological Anatomy, Royal College of Veterinary Medicine, Perouse, Italy, died on October 7, 1934, at Romentino (Novara). Dr. De Gasperi was the Italian member on the Permanent Commission of the International Veterinary Congresses, having succeeded Prof. Stazzi, Director of the Veterinary School at Milan, in May, 1933.

WILLIAM A. MABIE

Dr. W. A. Mabie, of Warsaw, Ind., died at his home, October 10, 1934. He had been in ill health for the past five years, but had been confined to his bed only a week before his death.

Born in Kosciusko County, Ind., May 16, 1857, Dr. Mabie was a graduate of the Chicago Veterinary College, class of 1891. A life-long resident of Kosciusko County, he was active in the life of his community. In addition to his professional activities, he also served two terms as sheriff of the County. Surviving are his window (née Catherine Secrist), two sons, a daughter, a sister, seven grandchildren and three great-grandchildren.

WILLIAM DANE GORDON

Dr. William Dane Gordon, of Morristown, Ind., died suddenly at his home, October 11, 1934, following a heart attack. Death was ascribed to aortic insufficiency.

Born in Rush County, Ind., December 4, 1891, Dr. Gordon was graduated from the Indiana Veterinary College in 1915. He enlisted in the Veterinary Corps during the World War and was commissioned a second lieutenant, October 4, 1917. He was ordered to active duty, July 2, 1918, and directed to report at Camp Greenleaf, Ga., for a course of instruction. He received his discharge papers, October 2, 1918, and returned to Rush County to resume his professional career. Nine years ago, he moved to Morristown and had since made his home there. During the past 12 years, he had served as an assistant state veterinarian of Indiana.

Dr. Gordon joind the A. V. M. A. in 1918. He was a captain in the Veterinary Reserve Corps and of the Morristown National Guard. He was also a member of the Masonic Lodge, at Crothersville, Ind. Surviving are his widow (née Rosa Bell Darnell), two sons and one daughter.

ERNEST ROSENTHAL

Dr. Ernest Rosenthal, of Omro, Wis., died suddenly on October 13, 1934, following a heart attack.

Born in Vinland, Wis., March 6, 1881, Dr. Rosenthal was a graduate of the Oshkosh (Wis.) High School and of the Oshkosh State Teachers' College. He attended the University of Wisconsin for a time before entering the Chicago Veterinary College, from which he was graduated in 1911. He was a member of Gamma Chapter of Alpha Phi Psi Fraternity, a member of the Omro Lodge of Odd Fellows and a volunteer member of the Omro Fire Department. He had practiced in Omro for the past 20 years. Surviving are his widow (née Pearl Kisner), one son, two daughters and six sisters.

JAMES THOMAS GLENNON

Dr. James T. Glennon, of Newark, N. J., died October 14, 1934, after a protracted illness.

Born in Newark, May 21, 1870, Dr. Glennon was educated in the public and parochial schools of his native city. He engaged in the stationery business for a time and then turned his atten-

tion to veterinary medicine. He studied under Dr. W. F. Harrison (Amer. '87), of Bloomfield, N. J., for three years before entering the New York College of Veterinary Surgeons, New York City. This institution later became a part of New York University. He was graduated in 1896 and was valedictorian of his class. In 1897, he took over the practice of Dr. Andrew G. Vogt (Amer. '84), of Newark, who had retired to accept the office of Receiver of Taxes for the City of Newark. Dr. Glennon held the positions of veterinarian to the Board of Public Works of Newark, to the Essex County Park Commissioner and, for a time, to the Newark Salvage Corps. Since 1899 he had been veterinarian to the Newark Fire Department, and had made an enviable reputation in that position.

Dr. Glennon joined the A. V. M. A. in 1899 and was a regular attendant at annual conventions for many years. He served as Resident Secretary of New Jersey (1903-07), as Chairman of the Committee on Finance (1913-15), as a member of the Committee on Necrology (1927-28), and as Second Vice-President for three terms (1929-32). He was a member of the Veterinary Medical Association of New Jersey, and was voted a life membership at the Fiftieth Anniversary Meeting of the Association, held January 4-5, 1934. Surviving Dr. Glennon is his widow (née Mary E. Quinn).

T. E. S.

FREDERIC SNOWDEN JONES

Dr. Frederic S. Jones, associate member of the Rockefeller Institute, Department of Animal and Plant Pathology, at Princeton, N. J., died at his home, October 19, 1934. He was 46 years old.

Born in Philadelphia, Pa., Dr. Jones attended local schools and the University of Pennsylvania. He was graduated from the Veterinary School in 1908 and for the next four years he was an instructor in the New York State Veterinary College at Cornell University. In 1913, he joined the staff of the Rockefeller Institute in New York and, three years later, was transferred to Princeton. In 1921, he was elected an associate member of the Institute.

Dr. Jones joined the A. V. M. A. in 1912. He was a member of the Committee on Veterinary Biological Products (1925-28). He was a member of the American Association for the Advancement of Science, the Association of Pathologists and Bacteriologists, the Society of Experimental Pathologists, and the So-

ciety of Experimental Biologists. Dr. Jones was a pioneer worker in the agglutination test for diagnosing pullorum disease in chickens.

Surviving Dr. Jones are his widow (née Maude Sinclair), one daughter and his mother.

PERSONALS

MARRIAGES

DR. HERMANN RUDOLPH SEIBOLD (U. P. '31), of New York, N. Y., to Miss Clara Bond Taylor, of New York, at New York, October 20, 1934.

DR. ROBERT O. BILTZ (U. P. '22), of Harrisburg, Pa., to Miss Catherine Mary Brightbill, of Hummelstown, Pa., at Hummelstown, October 13, 1934.

BIRTH

To DR. and MRS. C. L. BRIGGS, of Elizabethville, Pa., a daughter, Pauline Elnora, October 4, 1934.

PERSONALS

DR. J. H. JEFFERSON (Ont. '06), formerly of Sandusky, Ohio, has located at Kent, Ohio.

DR. H. H. STOVER (U. P. '34) has bought the practice of Dr. H. C. Terry at Langhorne, Pa.

DR. C. P. BROSE (O. S. U. '27) reports a change of address from Albany, N. Y., to Guilderland, N. Y.

DR. E. H. HOUCHIN (Wash. '24), of Hollywood, Calif., has bought the practice of Dr. P. H. Blickenstaff in Ventura, Calif.

DR. B. H. GRAY (McK. '15), of Windsor, Ill., has been appointed Will County Veterinarian and has removed to Joliet, Ill.

DR. I. C. MATTINGLY (St. Jos. '21), of Concordia, Kan., a former deputy state veterinarian of Missouri, has located at Anna, Ill.

DR. E. J. HART (McK. '11), of Pontiac, Ill., has been reappointed Livingston County Veterinarian. Dr. Hart has held this office for a number of years.

DR. J. T. BROWN (K. C. V. C. '15), of Belleville, Ill., has been reappointed Saint Clair County Veterinarian for another year by the County Board of Supervisors.

DR. N. F. RATERMAN (Cin. '12), of Fort Laramie, Ohio, was kicked by a horse recently while treating the animal. The blow was severe enough to loosen several teeth.

DR. HARRY WORCESTER (Cin. '07), of Middletown, Ohio, has received an appointment in the U. S. Bureau of Animal Industry and has been assigned to meat inspection in Chicago.

DR. F. L. RICHELIEU (Wash. '29), who is engaged in meat inspection for the California Department of Agriculture, has been transferred from Dinuba to Dixon.

DR. W. M. MARTINSEN (McK. '16), of Fremont, Ohio, suffered slight bruises in an automobile accident recently when his automobile sideswiped another car on State Route 19.

DR. ALBERT F. RANNEY (Corn. '32), formerly of Ithaca, N. Y., has accepted a position with the State Department of Agriculture, Montpelier, Vt., in charge of a Bang's disease laboratory.

DR. H. C. TERRY (Amer. '94), of Langhorne, Pa., recently retired from active practice, bringing to an end a professional career to which he has devoted himself during the past 40 years.

DR. E. WENDELL HOLDEN (Corn. '34) has entered general practice at Norristown, Pa. He plans to develop poultry practice in connection with his work with large and small animals.

DR. M. JACOB (U. P. '99), of Knoxville, Tenn., served as president of the Tennessee Valley Agricultural and Industrial Fair again this year. The Fair was held at Knoxville, September 24-29.

DR. G. W. WORRELL (St. Jos. '17) is now located at Stockton, Calif., where he is on meat inspection for the State Department of Agriculture. He was recently transferred from Dixon.

DR. P. H. BLICKENSTAFF (Wash. '23) recently sold his practice in Ventura, Calif., and has accepted a position in the State College of Washington, Pullman, as Assistant Professor of Veterinary Medicine.

DR. A. H. FRANK (Mich. '34), after practicing for a short time at Niles, Mich., has accepted an appointment in the U. S. Bureau of Animal Industry and is stationed in Chicago, Ill., on meat inspection.

DR. J. P. FOSTER (Ont. '00-McK. '08-Ont. '14), of Minneapolis, Minn., was a visitor to Lexington, Ky., in September, taking in the "Trots," visiting the breeding farms, and incidentally digging up a little more "history" of our veterinary colleges.

DR. E. W. NEWHALFEN (McK. '18), of Tiskilwa, Ill., together with his family, has located in Los Angeles, Calif., where he will assist in the operation of the western branch of the American Biochemic Company, of Peoria, Ill.

DR. GLENN H. SCOVILLE (Gr. Rap. '06), of Clarksville, Mich., was the victim of a robbery on September 28, when thieves entered his home and took $40 from the pockets of his trousers, and $5.35 from a bureau drawer.

DR. J. S. KOEN (K. C. V. C. '07) resigned his position as chief meat inspector with the Department of Public Welfare, Saint Louis, Mo., September 17, and has accepted re-instatement in the U. S. Bureau of Animal Industry. He has been assigned to meat inspection in Baltimore, Md.

DR. J. D. EASTWOLD (McK. '17), of Spooner, Wis., narrowly escaped losing his left eye recently, when he was hooked by a cow he was testing. Dr. Eastwold had finished fastening a tag to the ear of the cow, when she suddenly jerked her head loose and struck Dr. Eastwold just below the eye. The wound was severe enough to require several stitches.

DR. E. P. JOHNSON, (Mich. '25), of the Department of Zoölogy and Animal Pathology, Virginia Polytechnic Institute, recently was awarded the prize of $100 for the best piece of work published during the year in the field of poultry science. This is the first time the award has gone to a veterinarian. Dr. Johnson's paper, "The Etiology and Histogenesis of Leucosis and Lymphomatosis of Fowls," was published as Technical Bulletin No. 56 of the Virginia Agricultural Experiment Station.